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CONGENITAL HEART DISEASE: PART I. GENERAL SURVEY, METHODS OF INVESTIGATION AND RESULTS OF SURGERY AT THE ALFRED HOSPITAL.

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DURING the ten-year period from 1946 to 1955 inclusive, 883 cases of congenital heart disease have been studied by the Thoracic Surgical Unit and Cardio-vascular Diagnostic Service at the Alfred Hospital, Melbourne. In this paper it is intended to review our general experience, detail our method of approach, including the place of special investigations, and outline the indications for and results of surgical treatment. It is proposed in further papers to discuss common clinical groups and their diagnosis, with special reference to those lesions amenable to surgical correction.

GENERAL REVIEW OF SERIES.

In considering the incidence of the various anomalies in this series, it must be recognized that there is some selection of patients, with a bias to conditions with the possibility of surgical correction. Likewise the children examined tend to be in the older age groups. Complicated anomalies which occur in infancy, and which are com-

patible usually only with brief existence, do not form as large a proportion of this series as they may do in a children's hospital. We have not included in the series cases of primary pulmonary hypertension, as the aetiology of this condition is uncertain.

A summary of the cases investigated is given in Table I.

Diagnosis, derived by clinical methods, was confirmed as far as possible at operation in 414 cases, with special investigation (by cardiac catheterization or angiocardio-graphy or both) in 64 of those cases, and in 102 other cases in which no operation was undertaken. In 72 cases the final diagnosis was established at autopsy.

The cases remaining unclassified include eight cases in which special investigation failed to establish a firm diagnosis; in the remaining cases clinical diagnosis was uncertain, and special investigations were not performed, usually because the possible lesions were not in any event amenable to surgery, or because they were clearly too mild to warrant further investigation at this stage.

METHODS OF INVESTIGATION.

History.

Common modes of presentation of congenital heart lesions are the occurrence of cyanosis, the detection of a murmur, failure to thrive in infancy or poor exercise tolerance. A lesion may be suspected in the presence of other congenital anomalies, or because of a maternal history of rubella in the first trimester of pregnancy. In

the present series 41 patients had a maternal history of rubella; of these, 35 proved to have a patent *ductus arteriosus*, although in some the signs were atypical.

A history of recurrent bronchitis, occasionally with hæmoptysis, is suggestive of a large left-to-right shunt, especially with ventricular septal defect. On the other hand, a history of wheezing and inspiratory stridor, often associated with regurgitation of food, suggests the possibility of a vascular ring due to anomalies of the aortic arch.

In the cyanotic group, a history of squatting on exertion is highly suggestive of Fallot's tetralogy, only rarely being associated with other cyanotic congenital heart lesions.

It must be stressed that, with the increasing recognition of congenital heart disease, it is important not to overlook or label as congenital the characteristic lesions of rheumatic heart disease. This may occur particularly in patients who have no clear history of rheumatic fever and appear well clinically, or in whom the only finding is a pulmonary systolic murmur. Conversely, the error does arise of regarding as rheumatic heart disease congenital heart lesions complicated by bacterial endocarditis.

Physical Signs.

The diagnosis of the type of congenital heart lesion rests on the correlation of a number of pieces of evidence of the effects of that lesion on the cardio-vascular system. These include the presence or absence of cyanosis, the character of the arterial pulses, the jugular venous pulse, enlargement of the heart chambers, and auscultation of the heart.

Cyanosis.

In many instances there is no doubt as to the presence of central cyanosis, present at rest and associated usually with clubbing of the fingers, although in the first few months of life clubbing may be very difficult to detect. Particularly in Fallot's tetralogy, however, cyanosis may be absent at rest and appear only on exercise. Inspection of the tongue, mucous membranes and conjunctivæ is often of assistance in detecting mild degrees of central cyanosis. This is of particular value in the new-born infant, whose skin colour can be very variable and misleading, and is essential in differentiating central from peripheral cyanosis. The latter is often a functional phenomenon, but it may be associated with cardiac lesions with low output, particularly simple pulmonary stenosis. In some instances we have been unable to be certain on clinical grounds of the presence of central cyanosis; only occasionally have arterial oxygen estimations added further information to the clinical assessment.

The Arterial Pulse.

The arterial pulse is of value in the diagnosis of aortic stenosis, with its typical slow-rising character; in patent *ductus arteriosus*, particularly in infants, with its water-hammer quality; and in coarctation of the aorta, in which full pulses in the upper extremities and the neck are associated with a low-volume femoral pulse which, when compared with the radial pulse, shows easily discernible delay.

Jugular Venous Pulse.

The main value of observation of the jugular venous pulse has been the detection of the large abrupt "a" wave in simple pulmonary stenosis. The height of this wave gives some indication of the severity of this lesion, and in cases in which cyanosis is present it is of value in differentiating it from Fallot's tetralogy, in which the "a" wave is never striking.

Palpation of the Precordium.

Palpation of the precordium may give a valuable clue as to the ventricle predominantly affected by the lesion. A thrusting impulse at the apex indicates left ventricular hypertrophy, and a lifting impulse palpable in the third and fourth left intercostal space suggests right ventricular hypertrophy. It is important to stress that in patients with

thin chests and overacting hearts or in obese or barrel-chested patients due allowance must be made for normal variation in the precordial impulses. As will be pointed out below, confirmation of ventricular hypertrophy is best gained by electrocardiography.

Auscultation.

Auscultation reveals variation in the individual heart sounds, including absence, accentuation and delay, and murmurs which may be caused by the defects themselves, or by rapid flow through dilated chambers or vessels.

TABLE I.

Condition.	Male Patients.	Female Patients.	Patients Operated On.	Total.
Acyanotic:				
Coarctation of the aorta	55	28	61	83
Aortic and subaortic stenosis	18	6	3	24
Vascular rings			1	5
Dextrocardia (simple)			1	4
Ectopia cordis			1	1
Absent pericardium			1	1
von Gierke's disease			1	1
Congenital heart block			1	3
Simple pulmonary stenosis (acyanotic)	26	29	3	55
Idiopathic dilatation of pulmonary artery				5
Pulmonary incompetence				2
Ebstein's anomaly				2
Patent <i>ductus arteriosus</i>	48	137	161	185
Aortic-pulmonary septal defect			4	4
Ventricular septal defect	52	37	—	89
Atrial septal defect	28	30	5	63
Lutembacher syndrome				2
Partial anomalous pulmonary venous drainage				3
Single atrium				2
Atrial septal defect and ventricular septal defect				1
Atrio-ventricularis communis				2
Unclassified				34
Cyanotic:				
Fallot's tetralogy and pulmonary atresia (20 included)	131	85	163	216
Simple pulmonary stenosis (cyanotic)	6	7	7	13
Tricuspid atresia	8	7	6	15
Truncus arteriosus				2
Wilmmer's syndrome	15	16		31
Transposition of the great vessels	7	5		12
Ebstein's anomaly				3
Total anomalous pulmonary venous drainage				3
Single ventricle				1
Unclassified				21
Total cases			414	883

¹ Those cases in which a reversed shunt was present, proved to be via a patent *ductus arteriosus* or atrial septal defect, are not included in this group.

Comment.

Attempted diagnosis by consideration only of heart murmurs is likely to be unfruitful and at times misleading; consideration must be given to the total physiological effects of the lesion, as judged by information gained from all the points mentioned above. Nevertheless, we believe that the practitioner in his approach to the case will often have his attention caught first by the presence of a cardiac murmur. In Part II of this study we propose to discuss certain groups of cases in which the murmurs may be similar in character and distribution, and in which clinical differentiation may be difficult and confusing.

Instrumental Methods of Investigation.

The instrumental methods of investigation which we have employed as a routine are electrocardiography, radiological examination of the heart and, in many cases, phonocardiography. In special instances cardiac catheterization and angiocardiography have been used.

Electrocardiography.

The chief use of electrocardiography is to establish the presence of hypertrophy or dilatation of one or other ventricle, or of combined hypertrophy. In differentiating between right and left ventricular enlargement we have found this much more reliable than X-ray examination.

Radiological Examination.

Radiological examination of the heart, particularly fluoroscopic screening, is of help mainly in the following ways: (i) in assessing the general size of the heart, demonstrating its contour, which occasionally is characteristic, and perhaps assessing the size of the individual chambers, particularly the auricles, but rarely with certainty the ventricles; (ii) in assessing the size and character of pulsation of the pulmonary artery and of its peripheral branches; from this, deductions may be made about the pulmonary blood flow; (iii) in demonstrating some of the great vessel anomalies, especially those of the aortic arch.

Phonocardiography.

Phonocardiography is essentially an extension of clinical auscultation. We have used the method as a routine for the past three years, and have found it of great value. It provides essentially a method of recording in permanent form the heart sounds and murmurs, much as heard by the stethoscope, but timed against a reference tracing, so that the exact timing and relative intensity of heart sounds, and the shape and timing of murmurs can be established.

Help given by a phonocardiogram in the elucidation of the origin and significance of systolic murmurs will be detailed later in the discussion of clinical groups of cases.

The method is particularly valuable in the exact interpretation of the second heart sound, the character of which often gives a valuable guide as to pulmonary artery pressure and flow.

By recording heart sounds as they are registered at different areas simultaneously, by registering the dicrotic notch of the carotid pulse to identify the aortic element of the second heart sound, and by using the effect of inspiration in delaying pulmonary valve closure, both elements of the second heart sound can be studied. The pulmonary element in children is usually about as loud as the aortic element when recorded at the pulmonary area, although it is insignificant elsewhere. It may be delayed up to 0.08 second beyond the aortic element on inspiration, but returns to coincide with that sound on expiration. In pulmonary hypertension, the pulmonary element is often much accentuated. It may then be recorded more widely from the precordium; it usually follows the aortic element by a short but fixed interval, heard clinically as close splitting. In the left-right intracardiac shunts, even without pulmonary hypertension, the pulmonary element of the second sound is usually clearly recorded. In atrial septal defect, in which right ventricular ejection and pulmonary valve closure are delayed, the second heart sound is widely split, the pulmonary element following the aortic element by a relatively constant wide interval. It is not often accentuated in the pulmonary area, but in spite of this, is usually widely recorded. In ventricular septal defect and patent *ductus arteriosus* such wide splitting is not found, but the pulmonary element is usually loud enough for splitting of the second sound to be heard clearly on clinical examination. In Fallot's tetralogy the pulmonary element of the second heart sound is rarely discernible; hence the sound as recorded is purely aortic in origin and single. Nevertheless we have found in a number of cases that it is not a clear sound, but split up into a

number of coarse vibrations and often quite loud in the pulmonary area. This sound causes confusion clinically. However, the phonocardiogram shows that there is no true splitting into two separate components. When simple pulmonary stenosis is severe the situation is similar and no pulmonary element is recorded; the aortic element is often overlaid by the systolic murmur. However, in less severe cases the situation is different; the pulmonary element can frequently be recorded at the pulmonary area. It is often considerably delayed beyond the aortic element and is relatively soft, although this is less pronounced in mild cases, in which confusion may arise with mild examples of the left-to-right shunts. The pulmonary element of the second sound may reappear in severe cases after successful valvotomy. In aortic stenosis the aortic element is often normal in children, but in severe cases may be soft and even delayed beyond the normal pulmonary element.

The phonocardiogram is of value, too, in the examination of infants, in whom tachycardia and restlessness may make auscultation very difficult. A clear record of a few heart cycles may aid greatly in diagnosis. Thus the findings of a widely split second heart sound may point to a left-right interatrial shunt; a murmur thought to be systolic may be seen to pass through the second heart sound into diastole, and represent a typical though non-continuous Gibson murmur of patent *ductus arteriosus*. Greater precision in diagnosis may thus be attained.

Cardiac Catheterization and Angiocardiography.

Since cardiac catheterization and angiocardiography require admission of the patient to hospital, and for younger patients an anaesthetic, and involve a certain though small element of risk, they must be used with discretion. Each has its special uses and specific indications; both may be used in some cases, and even then may not always yield sufficient information for a complete diagnosis.

To decide which investigation will yield the most information, or, if both are necessary, which should be performed first, requires some discrimination and an understanding of their uses and limitations. In general these uses are as follows:

Cardiac catheterization will demonstrate: (i) the presence or absence of a left-right shunt and its situation; (ii) the presence or absence of pulmonary hypertension; (iii) the presence or absence of pulmonary stenosis, its site and, if simple, its severity. It may also demonstrate the presence of an abnormal communication, although it should be noted that the passage of a catheter through an opening (for example, *foramen ovale*) does not necessarily mean that the opening is functionally significant. However, it may provide sufficiently good evidence of the site of a right-left shunt, as for example in Fallot's tetralogy, in which catheterization of the aorta from the right ventricle demonstrates aortic overriding.

Angiocardiography will demonstrate: (i) the presence and site of a right-left shunt; (ii) the shape and disposition of the heart chambers and great vessels. It should be made clear, however, that the method will not give any direct estimation of pulmonary artery pressure, nor is the degree of opacification of the pulmonary vascular tree any true guide to the pulmonary blood flow under normal conditions. Thus in Fallot's tetralogy apparently adequate pulmonary vascular filling may be demonstrated, when clinically the patient is definitely disabled, and benefits considerably from systemic-pulmonary anastomosis. Angiocardiography, too, may fail to demonstrate the presence of pulmonary stenosis, and indeed the enormous post-stenotic dilatation of the pulmonary trunk may lead to an erroneous diagnosis of pulmonary hypertension. The presence or absence of pulmonary stenosis or of pulmonary hypertension is best established by cardiac catheterization.

In Table II are set out the investigations considered appropriate to particular lesions, if these tests are thought necessary. The indications and findings will be discussed in more detail in later papers dealing with the individual lesions.

The majority of patients in this series who have been submitted to operation have not had special investigation, since the clinical diagnosis of patent *ductus arteriosus*, coarctation of the aorta and Fallot's tetralogy may be established in the majority of cases by standard clinical methods, and the surgeons consider that technical problems of local anatomy (for example, in coarctation of the aorta) are best dealt with as seen at thoracotomy. On the other hand, in simple pulmonary stenosis cardiac catheterization has been considered necessary to estimate more accurately the severity of the lesion and to establish the site of stenosis to aid planning of the surgical approach. In cases of this condition when cyanosis is present, certain differentiation from Fallot's tetralogy, with demonstration of the interatrial shunt, has been considered essential.

Many patients not operated upon have undergone special investigation in order to establish a firm diagnosis and line of management, and to disprove operability.

TABLE II.

Condition.	Investigation.
Acyanotic:	
Coarctation	Angiocardiography.
Aortic stenosis	Angiocardiography.
Left-right shunt	Cardiac catheterization.
Simple pulmonary stenosis ..	Cardiac catheterization.
Cyanotic:	
Fallot's tetralogy:	
(i) Differential diagnosis from Eisenmenger syndrome	Cardiac catheterization.
(ii) Differential diagnosis from simple pulmonary stenosis	Cardiac catheterization and/or angiocardiography.
Eisenmenger syndrome ..	Cardiac catheterization, possibly angiocardiography.
Simple pulmonary stenosis ..	Cardiac catheterization and angiocardiography.
Pulmonary atresia	Angiocardiography.
Tricuspid atresia	Angiocardiography.
Ebstein's anomaly	Cardiac catheterization, possibly angiocardiography.
"Difficult"	Angiocardiography, then cardiac catheterization.

MEDICAL MANAGEMENT AND INDICATIONS FOR AND RESULTS OF SURGERY.

General Management.

It is important in the management of congenital heart lesions to allow the patients full freedom of activity. We do not feel that this can lead to any harm, and have seen more problems among those whose parents have persistently endeavoured to restrict their activity. These children should be encouraged to take part in sport at school, restriction being placed on severe competitive endeavour, as in long-distance running or rowing. As a practical consideration it has been found that, except for those with mild lesions, their capacity is rarely sufficient to enable them to reach competitive standard, and many, aware of their disability, do try to avoid sport, and occupy themselves more with hobbies.

Consideration of their training at school towards a suitable occupation is important, and considerable assistance can be gained by vocational guidance to ensure their placement when possible in satisfactory employment. It is advisable to avoid heavy manual work, or exposure to undue cold or to humid or damp conditions. The increasing insistence on insurance or medical examinations prior to employment has created a frustrating problem for those charged with the medical care of these patients, and it is to be hoped that some compromise will become available to make possible their employment in congenial occupations.

One problem in medical management is adequate protection during respiratory infections, with a longer period of bed rest than for normal children, and the use of penicillin in the treatment of the more severe infections. For those patients subject to recurrent respiratory infection we believe that sulphadiazine prophylaxis as used for rheu-

matic patients is of distinct advantage. We prefer the use of sulphonamide drugs to penicillin in prophylaxis, in view of the difficulties encountered in the treatment of bacterial endocarditis should a patient develop penicillin sensitivity.

Dental prophylaxis is important for these patients, and the parents must be warned to inform the dentist of the heart lesion, so that adequate penicillin cover can be used when extractions are necessary.

Finally, it is important to review these patients at regular intervals, to detect early evidence of cardiac failure and institute standard measures of treatment, to correct anaemia, faulty posture and nutritional defects at an early stage, and to reassess from time to time the patients' possible suitability for operation.

Indications for and Results of Surgery.

In this series of 883 patients, 414 were submitted to operation, the majority of lesions comprising patent *ductus arteriosus*, Fallot's tetralogy and coarctation of the aorta.

Coarctation of the Aorta.

Of 83 patients with coarctation of the aorta, 61 have been submitted to operation. Of these, two patients were inoperable, two had subclavian artery shunts, and three had aortic grafts. There were four deaths as a result of operation. Two other patients who had associated aortic stenosis died within two years after operation. In 43 of the 55 operable patients who survived operation, the blood pressure returned to normal post-operatively. In the remaining 12 cases it was reduced, although not to normal levels. Complete symptomatic relief was obtained in all patients except those with a significant complicating lesion.

In our opinion all patients with uncomplicated coarctation of the aorta up to the age of thirty years should be submitted to operation, the elective time being between the ages of ten and sixteen years. However, patients under the age of ten years should be operated on if there is evidence of increasing disability. Over the age of thirty years the risks of operation increase. Each case must be considered on its merits, and operation undertaken if there are progressive symptoms due to the coarctation. The oldest patient submitted to operation in this series was aged forty-six years.

There were six patients with associated aortic stenosis; of the three patients operated on, two died within two years. At necropsy it was considered that in neither case was the aortic valve suitable for aortic valvotomy. In the third case gross calcification of the aortic valve on X-ray examination was thought to contraindicate valvotomy. At present we doubt the advisability of operating on coarctation of the aorta in the presence of severe aortic stenosis.

There were 10 patients with associated aortic incompetence, all of whom have been submitted to operation without mortality. In one case in which severe incompetence was present it was not considered that operation produced any definite improvement. In our opinion patients with severe aortic incompetence should rarely be submitted to operation.

Vascular Rings.

Of five patients with vascular rings, three have been submitted to operation because of severe symptoms. In all these cases there was a right-sided aortic arch passing behind the oesophagus, with an obliterated ductus dragging the pulmonary artery hard back against the trachea in front. All patients were relieved by ligation of the obliterated ductus and underlying bands; in the youngest an aberrant subclavian artery, to which the ductus was attached, was divided as well. All three have remained symptom-free, although two of them still have a large filling defect in the back of the oesophagus on X-ray examination with a barium bolus.

We consider that all babies in whom the diagnosis is clearly established and who have any degree of disability from the lesion should be operated upon as soon as practicable, as intercurrent respiratory infection is a serious

hazard. One patient examined subsequent to this series died suddenly from respiratory infection while awaiting operation.

Patent Ductus Arteriosus.

Of 185 patients with patent ductus arteriosus, 161 were submitted to operation without mortality. In two cases closure was not successful and the ducts were divided at a subsequent operation. Of seven patients operated on prior to 1946, one died after operation.

The risks of operation are now minimal, and all patients under the age of forty years with uncomplicated patent ductus arteriosus should be submitted to operation. The oldest patient submitted to operation in this series was aged forty-five years. Unless operation is carried out, there is always the risk of bacterial endocarditis, and there is evidence to show that the life expectancy of these patients is limited. We consider that operation can be deferred until the age of three years, unless there is definite cardiac embarrassment. Four patients have been operated on in infancy, in all of whom the duct was found to be particularly large at operation. An urgent indication for operation is the complication of bacterial endarteritis. It is important that these patients have a full course of therapy prior to operation. In four such cases in this series operation was performed; in all a clinical cure was established.

Two patients were pregnant when examined. We do not consider that operation will usually be necessary during pregnancy, as there is rarely any pronounced degree of cardiac failure. There is, of course, the risk of infection of the ductus in the puerperium, and any puerperal sepsis must be rigorously treated.

In those cases of patent ductus arteriosus with associated pulmonary hypertension it has been our practice to divide the ductus, either if there is clinical evidence of a left-to-right shunt, as indicated especially by the character of the peripheral pulse at rest or on mild exertion, or if a left-to-right shunt is demonstrated on cardiac catheterization. In those cases in which no shunt is present or in which there is a right-to-left shunt with definite cyanosis, we do not consider division of the ductus to be of any value. There were five such cases in this series, in none of which has operation been undertaken.

Other Left-to-Right Shunts.

Aorto-Pulmonary Septal Defect.—There were four cases of aorto-pulmonary septal defect in this series, in all of which thoracotomy has been performed; in two cases repair of the defect was carried out, one with gratifying success. The other patient, an infant aged only six weeks, subsequently died of cardiac arrest.

Atrial Septal Defect.—In the atrial septal defect group operation has only recently been undertaken. Five patients have been submitted to operation. The first three were in *extremis* at the time of operation, and the defects were found to be unsuitable for repair. Of the next two patients, both had adequate repair of the septum. One unfortunately died of intercostal hemorrhage a week later. The other patient, a woman, aged fifty years, who had been in congestive cardiac failure for some years, has had an excellent result, and now has no evidence of failure and considerable improvement in exercise tolerance. This operation is becoming a safe procedure, but at the present time should be reserved for patients who have moderate disability. It is recognized that the onset of cardiac failure in childhood usually results from a large defect that is unlikely to prove suitable for blind closure and must await the development of open-heart surgery.

Simple Pulmonary Stenosis.

Of 55 acyanotic patients with simple pulmonary stenosis, only three, in all of whom the condition was severe, have as yet been submitted to operation. One of these patients had an infundibular resection. When they are first examined many patients with pulmonary stenosis have no disability. However, if the electrocardiogram shows the pattern of pronounced right ventricular preponderance, with inverted T waves in the right-sided chest leads,

cardiac catheterization to determine right ventricular pressure should be performed. Those patients with a resting right ventricular systolic pressure of over 100 millimetres of mercury should be seriously considered for operation. Patients with evident limitation of activity should be operated on without undue delay. It is considered that the present operation for infundibular stenosis is likely to be less satisfactory and more hazardous.

Cases of simple pulmonary stenosis with cyanosis are generally in the severe category. Of 13 patients who were cyanosed, six have been submitted to direct operation, with one death, the condition of the other patients being considerably improved.

Before this syndrome was clearly recognized, one patient had an anastomotic operation performed and died after operation. Of the remaining six patients, two died before operation could be carried out, one is a mongol, and at present the other three have not been considered sufficiently limited in activity to warrant operation.

Cyanotic Congenital Heart Disease.

Falot's Tetralogy (Including Pulmonary Atresia).—Of 216 patients with Falot's tetralogy, 163 have been submitted to operation; 144 had shunt operations, 85 having a Blalock type of anastomosis and 59 a Willis Pott's operation. Seven underwent direct valvotomy, and on 12 no definitive operation could be performed. Operative death occurred in eight of the shunt operations, in four of the direct valvotomies, and in six of the 12 cases in which the condition proved inoperable. One hundred and three patients who had a definitive operation have been followed up for a period of at least one year, up to a maximum of eight years. Of those 100 patients who had an anastomotic operation, 65 were considered to have a very good result, 21 a good result, nine a fair result, and five a poor result. Five of these patients have died since operation from various unrelated causes. Of the three patients who survived direct valvotomy, one had a very good and two had a good result.

We consider that operation should be performed on all patients with pronounced limitation of exercise tolerance, or with moderate limitation if it is persistent, particularly when squatting is frequent; on those with a considerable degree of cyanosis and associated polycythæmia; on patients with frequent dyspnoeic attacks; and finally when there is a persistent failure to thrive. On these results we have favoured the anastomotic operations, but open-heart surgery now offers the prospect of radical cure. It is desirable to defer operation until after the age of three years, provided the clinical features are not unduly severe. In two cases operation was performed at the age of six months, and 27 other patients were operated on before the age of three years. When patients are aged over three years, the lesion is reasonably mild and improvement is occurring, we prefer to defer operation until the limit of spontaneous improvement has been reached. A shunt operation was carried out on 10 adults, the oldest patient being aged thirty-four years. All these patients had very good results, with considerable diminution in cyanosis and increased exercise tolerance.

Tricuspid Atresia.—Of 15 patients with tricuspid atresia, six have been submitted to shunt operation, with one operative death. Two patients were operated on successfully in infancy, at the age of three months and four and a half months; the latter has maintained improvement for over six years. The other three patients have been considerably improved. The indications for operation are similar to those for Falot's tetralogy, although, as the results of operation may not be so satisfactory, we tend to defer operation when possible.

Apart from simple pulmonary stenosis with cyanosis, no other type of cyanotic congenital heart defect has as yet proved suitable for operation.

CONCLUSION.

The development of corrective surgery in patent ductus arteriosus and coarctation of the aorta, and of palliative surgery in Falot's tetralogy, gave great impetus to the

accurate diagnosis of these congenital cardiac defects, aided considerably by the elaboration of the techniques of cardiac catheterization and angiocardiography. A considerable degree of accuracy has now been attained in the clinical diagnosis of the lesions, and special investigations are relied on to a lesser extent. Newer techniques, such as hypothermia and the development of an artificial circulation, have now brought a much wider range of lesions within the scope of surgical correction, and no person with a congenital heart lesion, however hopeless this condition may appear, should be denied full investigation to determine accurately the nature of the lesion and to assess its operability. As will be seen from this survey, the results of surgery in the past have been gratifying, and the mortality has been remarkably low. The era of open-heart surgery is just beginning, and we can look forward with confidence to further major advances in this field.

CONGENITAL HEART DISEASE: PART II. COMMON CLINICAL GROUPS.

By H. B. KAY AND J. M. GARDINER,
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To facilitate discussion on the clinical diagnosis of congenital heart lesions, we propose to approach the problem on the basis of the murmurs audible and the presence or absence of cyanosis (Table I). The methods of investigation adopted have already been outlined (Officer Brown *et alii*, 1956).

In many instances the practitioner's attention will first be drawn to a case of suspected congenital heart disease by the presence of a murmur. The site and character of the murmur alone will rarely enable a clear-cut diagnosis to be made, but will place the case into a clinical group, within which differentiation may usually be effected by attention to the associated physical signs. It is only when the lesion is so mild that it causes virtually no appreciable physiological disturbance that clinical diagnosis must be reached solely on the site and character of the murmur. In this event phonocardiography may provide further differentiation, by attention to those points which will be detailed in the description of the various lesions.

CYANOTIC LESIONS.

Loud Basal Systolic Murmur.

The presence of a loud basal systolic murmur is invariably associated with a heart lesion or anomaly of the great vessels, and is commonly accompanied by a systolic thrill.

Aortic Stenosis.

A murmur situated maximally to the right of the sternum suggests aortic stenosis. In this lesion the murmur is conducted up to the neck vessels, the aortic second sound is diminished in more severe cases, a short early diastolic murmur may be audible down the left border of the sternum, and there is evidence of left ventricular enlargement. The peripheral pulse is often slow rising. It is important to palpate the femoral arterial pulse, as coarctation of the aorta is an occasional association. The electrocardiogram may be normal or show evidence of left ventricular enlargement.

X-ray examination may show evidence of left ventricular enlargement, and in some cases pronounced dilatation of the ascending aorta; the lung fields appear normal.

The phonocardiogram shows the murmur as diamond-shaped, with its peak before mid-systole, and a clear gap before the second heart sound, which in milder cases is normal. In severe cases this sound may be soft, even at the apex where it is normally recorded best; the sound becomes delayed, and may occur later than the normal pulmonary element. The murmur retains its relation to its own second sound, and may run up to or past the pulmonary element.

In children aortic stenosis is presumably congenital in origin, but in young adults it may be rheumatic. The

distinction can be made only in those with associated mitral valve disease, or if there is a clear history of rheumatic fever with subsequent development of the murmur.

When the murmur is situated maximally to the left of the sternum, further differentiation is made on the quality of the second sound in the pulmonary area.

Simple Pulmonary Stenosis.

If the sound is single, then the probable lesion is simple pulmonary stenosis. This diagnosis is confirmed by the finding of right ventricular hypertrophy, prominent "a" wave in the jugular venous pulse, and the X-ray appearance of post-stenotic dilatation of the pulmonary artery without pulmonary plethora.

Phonocardiographic examination shows the murmur to be roughly diamond-shaped, but the peak is normally in later systole, and the murmur runs up to the aortic element of the second sound. In severe cases the pulmonary element of the second sound is absent; in milder cases it may be recorded, may be softer than the aortic element, and may give rise to a fairly widely split second sound, which may be appreciated clinically.

The site of the murmur in valvular stenosis is usually in the second and first left intercostal spaces, but in the less common case of infundibular stenosis it is lower; in milder cases confusion may arise in the diagnosis from ventricular septal defect and occasionally aortic stenosis. Differentiation will depend on associated physiological disturbance, which will be minimal in mild lesions. In this group the phonocardiogram may be of assistance. In ventricular-septal defect the murmur is not usually diamond-shaped, except in the presence of pulmonary hypertension, when the pulmonary element of the second heart sound is loud. Precise diagnosis is made by cardiac catheterization, and occasional patients have been shown to have both pulmonary stenosis and ventricular septal defect combined.

Patent Ductus Arteriosus.

When the pulmonary second sound is definitely split, and the pulmonary element is loud and of the type heard in pulmonary hypertension, then the lesion may be a patent ductus arteriosus. Although the characteristic murmur in this condition is a continuous murmur, we have found that in occasional patients with a large ductus, especially in infants, the murmur may appear to be mainly or entirely systolic in timing. On phonocardiographic examination it can often be shown to extend into diastole. In this group of cases there are usually a wide pulse pressure and water-hammer pulse, and X-ray examination reveals pronounced pulmonary plethora. Recognition is important, for the degree of left-right shunt is considerable, and gross cardiac enlargement and even failure can ensue, whereas operation is curative.

Loud Parasternal Systolic Murmur.

Ventricular Septal Defect.

The most common lesion associated with a loud parasternal systolic murmur is ventricular septal defect. The murmur in this condition is usually maximal in the fourth left intercostal space, and is associated with a systolic thrill. Phonocardiographic examination shows it to be pan-systolic, and not diamond-shaped except in the presence of pulmonary hypertension. The second heart sound is usually narrowly but consistently split in the pulmonary area, the second pulmonary element being equal to or louder than the aortic element.

The electrocardiogram is normal in milder lesions; in more severe lesions there is evidence of combined left and right ventricular hypertrophy. On X-ray examination, the heart size and contour may be within normal limits in the milder cases; in more severe cases there is evidence of pulmonary plethora, the right and left ventricles are enlarged and the pulmonary trunk is prominent.

There are two variations from this typical clinical picture, which may give rise to difficulty in diagnosis.

First, the maximal site of the murmur may be in the third or even second left intercostal space, and differentiation must be made from pulmonary and aortic stenosis.

Secondly, in cases in which a larger defect is present, the systolic murmur may be less characteristic, being softer and more blowing; there is as a rule an associated mid-diastolic murmur at the apex, and evidence of considerable

TABLE I.

Type of Murmur.	Diagnosis.
Acyanotic Lesions.	
Loud basal systolic murmur	Aortic stenosis. Simple pulmonary stenosis. Patent ductus arteriosus (occasionally).
Loud parasternal systolic murmur.	Ventricular septal defect. Pulmonary stenosis (infundibular).
Soft systolic murmur, basal or parasternal.	Functional: sternal depression. Atrial septal defect. Partial anomalous pulmonary venous drainage. Idiopathic dilatation of pulmonary artery. Primary pulmonary hypertension.
Systolic murmur at the apex	Coarctation of the aorta. Mitral valve disease.
Continuous murmur	Patent ductus arteriosus. Aorto-pulmonary septal defect. Venous hum. Coarctation of the aorta. Pulmonary arterio-venous aneurysm.
Diastolic murmur:	
(a) Pulmonary area	Pulmonary incompetence. Atrial septal defect. Pulmonary hypertension.
(b) Parasternal	Sternal depression. Atrial septal defect. Aortic incompetence.
(c) Mitral	Patent ductus arteriosus. Ventricular septal defect. Lutembacher's syndrome.
No murmurs	Anomalies of the aortic arch.
Cyanotic Lesions.	
Harsh systolic murmur at left sternal edge.	Fallot's tetralogy. Simple pulmonary stenosis with reversed shunt. Tricuspid atresia.
Soft systolic murmur:	
(i) Second sound at pulmonary area single	Fallot's tetralogy. Tricuspid atresia. Pulmonary atresia.
(ii) Second sound at pulmonary area loud and split.	Eisenmenger's syndrome. Transposition of the great vessels.
Diastolic murmur:	
(i) Continuous	Anastomotic circulation: bronchial arteries or patent ductus arteriosus.
(ii) Pulmonary diastolic murmur.	Pulmonary arterio-venous aneurysm. Eisenmenger's syndrome.
Variable murmurs	Ebstein's anomaly.
No murmurs	Pulmonary atresia. Total anomalous pulmonary venous drainage.

cardiac enlargement with left and right ventricular hypertrophy. In this group there is radiological evidence of pronounced pulmonary plethora. Differentiation from similar cases due to other large left-to-right shunts, particularly patent ductus arteriosus with atypical signs, may prove difficult. The character of the pulse may give a lead to the possibility of a patent ductus, but diagnosis can be established with certainty only on cardiac catheterization, as occasional patients with ventricular septal defect have associated aortic incompetence.

Soft Systolic Murmur, Basal or Parasternal.

The basal or parasternal soft systolic murmur is commonly functional in origin, associated with an over-acting heart, anaemia or chest deformity.

Sternal depression or flattening of the anterior chest wall with or without associated scoliosis may give rise to findings erroneously regarded as due to congenital heart lesions. The systolic murmur is often superficial in quality, and may disappear in the upright posture or on deep inspiration. The pulmonary second sound is often loud and split. The electrocardiogram may show an rSr¹ pattern in the right precordial leads, but is otherwise normal. The radiological appearance may give rise to some difficulty, as the pulmonary trunk appears dilated, although it is commonly more transparent than that seen in congenital heart lesions causing pulmonary artery dilatation and its pulsation is normal. With grosser degrees of sternal depression, the heart appears enlarged in the postero-anterior view, but oblique views reveal no overall cardiac enlargement.

Difficulty arises occasionally when patients with congenital heart lesions, particularly mild atrial septal defect and pulmonary stenosis with little physiological disturbance, have associated chest deformity. The diagnosis can often be established only by special investigation.

Atrial Septal Defect.

The commonest congenital heart lesion to give rise to a soft basal or parasternal systolic murmur is atrial septal defect. As the murmur is not in itself characteristic, the diagnosis depends particularly on evidence of associated physiological changes. There is commonly only a soft to moderate systolic murmur which may be superficial in quality, similar to the murmur of sternal depression; phonocardiographically it occupies early and middle systole, and fades off before the second heart sound. However, in association there is evidence of right ventricular hypertrophy and dilatation, as shown by vigorous pulsation in the third and fourth left intercostal spaces. There is wide splitting of the second heart sound in the pulmonary area, and occasionally there is either a pulmonary diastolic murmur or a superficial mid-diastolic murmur audible towards the apex. This lesion is one of the few congenital heart lesions that may be associated with auricular fibrillation or flutter. The electrocardiogram shows a right bundle branch block pattern, either partial or complete. On X-ray examination there may be either striking dilatation of the pulmonary trunk and its main branches, with associated plethora and cardiac enlargement, or main pulmonary artery dilatation only, with little or no cardiac enlargement. There is frequently evidence of right auricular enlargement, but X-ray examination with a barium bolus reveals no left auricular enlargement. In our experience it is not common for atrial septal defect to be associated with a loud systolic murmur or an associated thrill. If a systolic thrill is palpable, we suspect associated pulmonary stenosis, unless a very large shunt is present.

Lutembacher's Syndrome.

Atrial septal defect is occasionally combined with organic mitral stenosis, known as Lutembacher's syndrome. In this condition the characteristic mid-diastolic murmur of mitral stenosis is heard at the apex. On X-ray examination, left auricular enlargement may be demonstrated and occasionally mitral valve calcification. It should be stressed that only a few of the patients with atrial septal defect and a mid-diastolic murmur audible at or near the apex prove to have organic mitral stenosis. Of 60 patients with atrial septal defect in the present series, only two had proven mitral valve disease. Exact diagnosis of the lesion can be established only by cardiac catheterization, with demonstration both of the atrial defect and of stenosis of the mitral valve as indicated by a significant diastolic pressure difference across the mitral valve.

Anomalous Drainage of Pulmonary Veins.

Physiological effects, and consequently physical signs, which are similar to those of atrial septal defect are encountered in partial anomalous drainage of the pulmonary veins into the right auricle. In this lesion a left-right shunt into the right auricle will be demonstrated by cardiac catheterization, as in atrial septal defect; proof

of abnormal pulmonary venous connexion depends upon catheterization of the abnormal veins. An attempt has been made to differentiate this condition from atrial septal defect by the use of dye injection techniques, but the findings are not always clear cut, and in any event the two lesions may occur in association.

Idiopathic Dilatation of the Pulmonary Artery.

Idiopathic dilatation of the pulmonary artery may be associated with a similar soft basal systolic murmur; other clinical features are normal, but X-ray examination reveals dilatation of the pulmonary trunk, without associated plethora. Occasionally a fibrotic lesion on the left lung, causing traction of the left pulmonary artery, or hilar gland masses may give rise to confusion. In some instances doubt may exist as to the possibility of an underlying cardiac lesion, particularly atrial septal defect. In these circumstances special investigation, usually cardiac catheterization, will be necessary to exclude any cardiac lesion.

Primary Pulmonary Hypertension.

Cases of primary pulmonary hypertension have not been included in this survey of congenital heart disease, as it is not yet certain that this condition is congenital in origin. During the period of this survey six proven cases have been encountered. The lesion should be considered in this group. It is differentiated by the presence of a loud split second sound in the pulmonary area, occasionally the murmur of pulmonary incompetence, evidence of strong right ventricular hypertrophy and on X-ray examination the absence of pulmonary plethora, with clear peripheral lung fields. Confirmation of this lesion is established by cardiac catheterization.

Systolic Murmurs Maximal at the Apex.

Systolic murmurs maximal at the apex are not commonly seen in congenital heart lesions, and usually indicate acquired heart disease, in particular rheumatic mitral valve disease. However, they may appear in association with coarctation of the aorta. It should be stressed that in coarctation of the aorta a systolic murmur may be audible in any area of the precordium, and posteriorly. Consequently in all patients with suspected acyanotic congenital heart disease, coarctation of the aorta must be excluded. In this condition there is usually some degree of hypertension in the upper extremities; the femoral pulse is of low volume, and its peak is delayed when compared with that of the radial pulse. Other features are the collateral circulation over the upper part of the chest, particularly along the scapular borders, and on X-ray examination the absent aortic knuckle and notching of the ribs.

Continuous Murmurs.

Patent Ductus Arteriosus.

Situated in the first and second left intercostal space is the continuous murmur of a patent *ductus arteriosus*, first described by Gibson in 1900. The murmur starts after the first heart sound, increases in *crescendo* up to the second heart sound and passes through into diastole, where it fades again. In mild cases it may not be strictly continuous, but retains this characteristic timing. The phonocardiogram will demonstrate this unequivocally, and will aid in its differentiation from the systolic murmurs mentioned earlier, especially in infants, and from the to-and-fro murmurs of pulmonary or aortic incompetence. In the latter, rather than a *crescendo*, there is a clear gap before the second heart sound. The associated features of patent *ductus arteriosus* have already been described.

A similar murmur audible maximally in the second and third intercostal spaces, although occasionally heard with a patent *ductus arteriosus*, suggests the presence of an aorto-pulmonary septal defect.

It is important not to confuse this type of murmur with that of a venous hum, which is not persistent, disappearing either with postural change or with pressure on the root of the neck blocking venous return.

Occasionally in coarctation of the aorta the anastomotic circulation may give rise to a continuous murmur. Other causes of a continuous murmur in the chest are pulmonary arterio-venous aneurysm, and the bronchial artery anastomotic circulation in pulmonary atresia. These lesions are usually cyanotic, but in occasional cases central cyanosis may not be evident.

Diastolic Murmurs.

In the pulmonary area an early diastolic murmur is usually due to pulmonary incompetence, and is distinguished from an aortic diastolic murmur by the fact that it commonly radiates laterally above the nipple, that it is usually inaudible to the right of the sternum, and is associated with the features of right cardiac enlargement and pulmonary hypertension. If these last-mentioned features are lacking, distinction from the murmur of aortic incompetence may be very difficult, and in our experience most older children with an early diastolic murmur heard at the left sternal edge have rheumatic aortic incompetence.

A short superficial diastolic murmur may be heard along the left sternal border in the presence of pronounced sternal depression; there is usually an associated "scratchy" systolic murmur.

A mid-diastolic murmur may be audible at the apex in those congenital heart lesions associated with considerable left-to-right shunts; in fact it is regularly found in association with patent *ductus arteriosus* and ventricular septal defect with large shunts. The murmur is similar to that of rheumatic mitral valve disease; differentiation must be made on the associated findings. The diastolic murmurs heard in cases of atrial septal defect have already been discussed.

No Murmurs.

Occasionally acyanotic heart lesions may have no associated murmurs. This may occur in coarctation of the aorta, or in other anomalies of the aortic arch. Of particular importance are those anomalies producing a vascular ring, with its characteristic history of wheezing, regurgitation and dysphagia from early infancy. In the commonest form, due to a persistent right aortic arch, X-ray examination with a barium bolus will reveal a zone of compression in the oesophagus, with displacement forward and to the left; when necessary, tracheography and bronchoscopy confirm the presence of tracheal compression. Further information as to the exact anatomy can be obtained by angiocardiography.

CYANOTIC LESIONS.

The main practical consideration in the diagnosis of cases of cyanotic congenital heart disease is to determine whether or not the case falls into the group associated with low pulmonary artery pressure and flow. It is patients in this group who can usually be helped by operation, either by systemic-pulmonary anastomosis as in Fallot's tetralogy, pulmonary atresia and tricuspid atresia, or by direct operation in cases of simple pulmonary stenosis and some cases of Fallot's tetralogy. In these lesions the significant common features are a single second heart sound in the pulmonary area, which is not unduly loud, and diminished pulmonary vascular markings on X-ray examination. No lesion in which there is high pulmonary pressure or high pulmonary flow is at present operable. The common findings in this group are a very loud and often clearly split second sound in the pulmonary area, and on X-ray examination a dilated pulmonary trunk, often with associated pulmonary plethora.

Harsh Systolic Murmur at Left Sternal Edge.

Fallot's Tetralogy.

Fallot's tetralogy is the commonest lesion of the operable group in which there is a harsh systolic murmur at the left sternal edge. In this condition there is a history of undue dyspnoea on effort which is commonly relieved by squatting, of dyspnoea or cyanotic turns, and of cyanosis and clubbing appearing early in life. Occasionally the last-mentioned features are not evident until later childhood,

and in one case in this series neither clubbing nor cyanosis was present even on effort, although at necropsy at the age of twenty-two years the typical lesion of Fallot's tetralogy was present. The systolic murmur is usually only moderate in intensity, and not often accompanied by a thrill; it is usually maximal in the third left intercostal space, but occasionally is more intense, and heard equally well in the second and first intercostal spaces. The second sound at the pulmonary area is single or "split up" (that is, not clearly split into two elements), although it may on occasion be loud. Although the jugular venous pulse may show an "a" wave, this is of normal size and not the prominent wave seen in simple pulmonary stenosis. Electrocardiographic evidence of right ventricular enlargement is present. On X-ray examination the characteristic features are the "sabot" appearance of the apex, usually a diminished pulmonary artery segment, and ischemic lung fields.

The aorta was right-sided in 37 of 216 cases in the present series.

Variations from the characteristic radiological appearance have been encountered. In some cases there is a more rounded apex; in others a fullness below the true pulmonary artery segment represents an infundibular chamber. Slight fullness of the pulmonary trunk was seen in some patients with valvular stenosis, but was never in our experience of the degree seen in simple pulmonary stenosis. The lung fields may not have the typical ischemic appearance in some cases, particularly in younger children, in the examination of whom difficulty may arise in taking films in full inspiration; shadowing above the hilar regions may also occur when there is pronounced bronchial artery anastomosis.

The phonocardiogram usually shows a moderate systolic murmur commonly in the third and fourth intercostal spaces; it is pan-systolic, but often tends to fade off a little before the second heart sound. This is in contrast to the murmur in simple pulmonary stenosis, which has a diamond shape and late systolic peak, and runs up to the aortic element of the second heart sound. The second heart sound in Fallot's tetralogy may be loud, and this may lead to confusion, but this sound is always single, and on phonocardiography is shown to be due to the aortic element of the second sound; a pulmonary element is rarely recorded, but a soft delayed sound has been found occasionally in this series.

In the majority of cases, the clinical features have been sufficiently characteristic to enable an accurate diagnosis to be established; but there are two groups in which special investigation is considered to be important.

In the first group there is little doubt that pulmonary stenosis is present, for the murmur is particularly loud and accompanied by a thrill, and the second sound is single and relatively soft. However, the murmur is higher than that usually heard in Fallot's tetralogy, extending to the second and first intercostal spaces; the electrocardiogram shows more pronounced *S-T* segment and *T* wave changes in right-sided chest leads than is usual in this condition, and on X-ray examination the pulmonary artery is rather prominent. In these cases one may suspect the presence of simple pulmonary stenosis with reversed shunt between the auricles. Cardiac catheterization and possibly angiocardiology are needed to make the distinction with certainty.

The second group, on the other hand, includes cases in which there are a soft or insignificant systolic murmur at the sternal edge, a loud and rather ringing or "split-up" second heart sound, and pulmonary vascular markings which are a little heavier than is usual. This group must be distinguished from cases of the Eisenmenger syndrome, which is not an operable lesion; the distinction can be made with certainty on cardiac catheterization.

Simple Pulmonary Stenosis with Reversed Shunt.

Simple pulmonary stenosis with reversed shunt, usually interatrial, should be suspected when there is no history of squatting and less increase in symptoms and cyanosis

on effort; when there are a harsh systolic murmur and thrill in the first and second left intercostal space and the second sound in the pulmonary area is widely split with a soft second element; when there is a prominent "a" wave in the jugular pulse; when the electrocardiogram shows a strong pattern of right ventricular preponderance with *T* wave inversion in the precordial leads; or when the X-ray examination does not reveal a characteristic "sabot" apex and there is a full and dilated pulmonary trunk.

It is of paramount importance to distinguish this lesion from Fallot's tetralogy, as the performance of a shunt operation on patients in this group is often fatal. When the distinction is in doubt on clinical grounds and operation is contemplated, special investigation should be carried out. Angiocardiography will demonstrate the route of right-to-left shunt. In this lesion the dye passes from the right to the left auricle in early films and is visualized in the aorta slightly later, whereas in Fallot's tetralogy the dye passes directly from the right ventricle into the aorta without opacification of the left heart chambers.

Tricuspid Atresia.

The clinical features of tricuspid atresia are similar to those in Fallot's tetralogy, but the diagnosis is readily established by evidence of left ventricular hypertrophy on electrocardiographic examination. The diagnosis is confirmed by angiocardiography.

In some cases of tricuspid atresia there is compensating transposition of the great vessels. One patient of the present series was considered to have this double anomaly.

Soft Systolic Murmurs.

There are two main groups of cyanotic congenital heart conditions in which the murmur is soft.

Fallot's Tetralogy and Tricuspid Atresia.

In the first group the second sound in the pulmonary area is single; this includes some cases of Fallot's tetralogy and tricuspid atresia. In pulmonary atresia, which has many features similar to Fallot's tetralogy, there may be only a soft murmur or no murmur. However, the continuous murmur of an anastomotic circulation may be audible, often heard widely beneath the clavicles and posteriorly. In these cases, X-ray examination usually reveals a strikingly hollow pulmonary artery segment, and a prominent aorta; the pulmonary arteries may be present or absent. Diagnosis is confirmed by angiocardiography, which may demonstrate the presence of pulmonary arteries large enough to enable a shunt operation to be performed. When this method fails to demonstrate the size of the pulmonary arteries, operation is still justified, as suitable arteries for anastomosis may be present.

Eisenmenger Syndrome and Transposition of Great Vessels.

In the second group the second sound in the pulmonary area is loud and split, indicative of pulmonary hypertension, or of increased pulmonary blood flow. This group comprises cases of the Eisenmenger syndrome and transposition of the great vessels.

The Eisenmenger syndrome includes both the true Eisenmenger complex, with the aorta overriding the right ventricle, and examples of pulmonary hypertension with high pulmonary resistance leading to right-to-left shunt through a patent *ductus arteriosus*, aorto-pulmonary septal defect, ventricular or atrial septal defects. Although some of these patients do not become cyanosed until early adult life, others may be cyanosed from early infancy. Often the cyanosis is only slight, as is the functional incapacity; squatting and dyspnoeic turns are rare, but there is a tendency to haemoptysis and recurrent pulmonary infection. Other signs of pulmonary hypertension include the presence of a pulmonary diastolic murmur, palpable pulmonary artery pulsation and right ventricular enlargement. On X-ray examination there is dilatation of the pulmonary trunk and often of the main pulmonary arteries, with or without associated pulmonary plethora.

In the differentiation from cases of low pulmonary artery pressure, from the standpoint of possible surgical treatment the important investigation is the proof of pulmonary hypertension by cardiac catheterization. Demonstration of the site of the shunt, either by catheterization or by angiocardigraphy, is of less practical importance. Deductions about the presence of pulmonary hypertension from the appearance of the pulmonary vessels at angiocardigraphic examination may be quite misleading.

Transposition of the great vessels is commonly associated with deep cyanosis from early life; the heart may be normal in size shortly after birth, but rapidly enlarges. The murmurs are variable, there is right ventricular enlargement, and on X-ray examination there is considerable enlargement of the heart with considerable pulmonary plethora in most cases. In some cases the typical narrow vascular pedicle is seen in the antero-posterior projection, in others the transposed aorta forms a prominent left upper border of the heart shadow, whereas in still others no characteristic basal silhouette is seen.

Angiocardiographic examination will demonstrate the origin of the aorta anteriorly from the right ventricle.

Diastolic Murmurs.

Occasionally diastolic murmurs are audible in cyanotic congenital heart disease.

A continuous murmur consistent with the presence of an anastomotic circulation may be due either to a patent *ductus arteriosus* acting as a compensatory lesion, when it is characteristic in its site and loudness, or to an anastomotic circulation through the bronchial arteries, softer than the murmur of a patent *ductus arteriosus* and frequently audible in both the right and left upper quadrants of the chest, anteriorly and posteriorly. It is usually associated with pulmonary atresia, but may also be found in tricuspid atresia and occasionally in Fallot's tetralogy. Care must be taken to exclude a pulmonary arterio-venous aneurysm.

The occurrence of a pulmonary diastolic murmur in Eisenmenger's syndrome has already been mentioned.

No Murmurs.

No murmurs may be audible over the precordium in patients with pulmonary atresia, or in patients with total anomalous pulmonary venous drainage. We have encountered only three cases of the latter lesion in this series. The signs included relatively slight cyanosis, features of pulmonary hypertension and X-ray appearance of pulmonary plethora. In one case in which drainage was into a vertical vein joining with the left innominate vein, a characteristic cottage-loaf appearance was seen on X-ray examination.

Ebstein's Anomaly.

One lesion which has variable features on auscultation is Ebstein's anomaly of the tricuspid valve; these patients may be acyanotic, but more often there is a shunt from the right to the left auricle. There may be a history of paroxysmal arrhythmia; there is often little disability in contrast with the considerable enlargement of the heart. On auscultation, systolic and diastolic murmurs and triple or quadruple rhythms have been observed. The electrocardiogram characteristically shows a long P-R interval and a right bundle branch block pattern which may be bizarre. X-ray examination reveals considerable enlargement of the heart, due to the enormous dilatation of the right auricle and part of the right ventricle. The lung fields are rather ischaemic. The diagnosis can usually be established on the clinical features, and as special investigation has been attended by a relatively high mortality in this group, owing to the occurrence of arrhythmias, it should be avoided if possible. Occasionally the condition may be difficult to distinguish from pulmonary stenosis; in Ebstein's anomaly cardiac catheterization will demonstrate low pressures in the pulmonary arteries and throughout the right side of the heart (Gardiner and Kay, 1956).

CLINICAL GROUPS IN INFANTS.

In infancy accurate diagnosis of the type of congenital heart lesion is less easy to attain, partly because a number of lesions occurring at this age are complicated and bizarre, and partly because physical signs are more difficult to elicit and interpret.

Auscultation tends to be less helpful than in the examination of older children. Functional murmurs are common. Heart sounds and murmurs are harder to localize and time accurately; the phonocardiogram is of real assistance in this regard. Electrocardiography is also less helpful, because of the physiological right ventricular dominance which exists at this stage, and the wide range of normality. The cardiac silhouette, too, as viewed by X-ray examination, has a wide variation in normal infants. Fluoroscopic examination is important, in demonstrating the size and contour of the heart, and the vascularity of the lung fields, which may be judged at all phases of respiration, in particular on full inspiration.

Infants with congenital heart disease present in several groups, as follows.

1. In the first group are the infants with cyanosis. This may be severe and generalized, but lesser grades may be seen only in the lips and tongue. Respiratory causes must be excluded. If central cyanosis is present, the practical point is the same as for older children—the differentiation of Fallot's tetralogy and other conditions with low pulmonary artery pressure and flow from conditions with high pulmonary pressures. The commonest lesion in the latter group at this age is transposition of the great vessels. Heart sounds, murmurs and the electrocardiogram will be of little help, unless the last-mentioned shows strong left axis deviation, suggesting tricuspid atresia. Determination of the vascularity of the lung fields by X-ray examination is the crucial point; it shows ischaemic lung fields in the first group, but as a rule pulmonary plethora in the second. In transposition of the great vessels the heart size on X-ray examination may be normal shortly after birth, but the heart rapidly enlarges in the first few weeks, and plethora may be intense. At present this type of lesion is inoperable. But if the lung fields are ischaemic and the heart is sabot-shaped or boot-shaped, and if cyanosis is increasing or there are cyanotic attacks, the diagnosis is almost certainly Fallot's tetralogy. Confirmation may be obtained by angiocardiography, or it may be thought advisable to proceed directly to thoracotomy. We have obtained good results, lasting for at least six years, by aorto-pulmonary anastomosis in infants as young as three months old.

2. The second group comprises those acyanotic, often pale infants with large hearts, who thrive poorly and are subject to repeated respiratory infections. They may die in cardiac failure at an early age. The majority of these infants have a lesion with a large left-to-right shunt, the commonest being ventricular septal defect. These patients have in common a hyperactive heart, which is enlarged clinically and radiologically, with pronounced pulmonary plethora. Often there is a loud mid-diastolic murmur at the apex. In ventricular septal defect there is usually a loud, harsh, systolic murmur heard best low down near the epigastrium, but occasionally it is higher in position or relatively soft. The important point to recognize is that a large patent *ductus arteriosus* can produce a very similar picture. The murmur is usually higher, in the pulmonary area and above, and mainly systolic in timing in these cases. There may not be the typical Gibson murmur running into diastole. However, the pulse pressure is almost invariably wide, and the pulse has a water-hammer quality. Recognition is vital, for the condition is curable by operation, but if it is untreated it may lead to progressive disability or death in cardiac failure. Proof of the lesion may be obtained by cardiac catheterization or more directly by thoracotomy. Catheterization in this series was performed more often in cases of ventricular septal defect, for certain exclusion of an operable lesion.

Other severe acyanotic lesions may give rise to a somewhat similar picture, with possible cardiac failure; these

include aortic stenosis, simple pulmonary stenosis and coarctation of the aorta. In these the signs of a left-right shunt are absent, and the characteristic features of the lesion should lead to correct diagnosis. In this group also must fall cases of glycogen storage disease and of fibroelastosis. No proven cases of the latter have been encountered in this series. The lesion is uncommon, but is being increasingly recognized. It may affect the endocardium of the chambers or valves of any part of the heart, and often leads to rapid and unexpected cardiac failure. Ante-mortem diagnosis is very difficult, but the rapidly progressive course, bizarre signs and electrocardiographic changes of "ventricular strain" may give clues as to the condition.

A much more common problem in relation to this group of infants is the differentiation of infants without organic congenital heart disease who present with suggestive symptoms, such as difficulty in feeding, tachycardia and easy fatigue. On examination of the infants a systolic murmur may be audible, but is usually soft and variable. Occasionally it may be louder and more persistent. Certain exclusion of a congenital heart lesion may be difficult, but will rest largely on the demonstration of a normal electrocardiogram, and normal findings on fluoroscopic screening of the heart.

3. A third group comprises the infants with loud cardiac murmurs, and possibly slight cardiac enlargement, but no definite symptoms. The nature of the murmur will usually make it clear that a congenital lesion is present, but exact anatomical localization is not so important at this stage, and can wait until the child grows.

CONCLUSION.

We have attempted to present an account of the commoner congenital heart lesions and their clinical differentiation, taking as a basis the outstanding physical signs of cyanosis and heart murmurs. It should be realized that not all cases encountered will fit neatly into the clinical groups outlined. Special investigation may be necessary for final differentiation, but should generally be reserved for doubtful cases in which a firm diagnosis is essential in deciding management, particularly when there is the possibility of surgical correction. We believe that in the majority of cases, including most of those in which operation is contemplated, diagnosis can be established by standard clinical methods.

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DERMOIDS, DIMPLES AND SPINAL MENINGITIS.

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DERMOIDS associated with spinal cord and *cauda equina* lesions are still of sufficient rarity to warrant reporting. In addition, they are not infrequently combined with other congenital malformations, amongst which *spina bifida occulta* is readily recognized on an X-ray film but is rarely of clinical importance, whilst a small dimple on the skin of the back is easily overlooked, yet may be of considerable significance. These dimples, which are often surrounded by pigmented or hirsute patches, may mark the cutaneous opening of an epithelium-lined track, which discharges intermittently upon the surface and acts as a route of infection to the spinal meninges. Such infection can result in abscess formation, in recurring meningitis or even in chronic meningitis sometimes mistaken for a tuberculous infection.

Historical Survey and Nomenclature.

In studying the literature one is struck by the somewhat varied terminology, which can lead to confusion. Cruveilhier (1829) first referred to "*tumeurs peries*" of the central nervous system in his report of three intracranial tumours resembling mother-of-pearl. These so-called pearly tumours have appeared on rare occasions in the spinal cord as well as in the cranial cavity, and they are, in fact, epidermoids—that is to say, they grow from ectodermal inclusions which have produced solely superficial epithelial structures, and the shining appearance is the result of epidermal desquamation over a prolonged period. Should the accidentally included ectodermal cells have produced hair follicles and sebaceous glands, there occurs a collection of grumous, white material mixed with hairs, as in the case reported. Such tumours are dermoids, and it may be noted that under the heading of pearly tumour some authors refer to both epidermoids and dermoids, though the majority apply it only to the former. Teratomata are those tumours which contain derivatives of all the primitive embryonic layers—ectoderm, mesoderm and endoderm; but there is a sub-group recognized by some, the teratoid tumours, which contain the representatives of two germinal layers.

To revert to the dermoids, they have been called cholesteatomata, a term also employed to describe at least two other unrelated conditions—to wit, the chronic excavation of the middle ear and petrous temporal bone associated with chronic *otitis media* and perforation of the drum, and certain tumours appearing in the vicinity of the pituitary fossa in which cholesterol crystals are found.

Finally, other terms are encountered which help confuse the issue still further; but their use is confined to the older writings and they do not bear discussion in a short paper.

The simplest classification of the tumour types is based on their embryonic derivation, and consists of the following: true teratomata, in which all three germinal layers are represented; and the dermoids and epidermoids, which arise only from the ectodermal derivatives.

Report of a Case.

B., a male patient, aged twenty-six years, was admitted to Saint Vincent's Hospital, Sydney, on September 28, 1953. He said that he had been in perfect health until one month previously, when he was wrestling with a friend for exercise. He fell to the ground, and found that he could not rise or stand on his feet, and complained simultaneously of pain in the lower lumbar region. In the course of the ensuing month, paresthesia developed in both his feet, and he suffered from shooting pains starting in his right knee and passing up the anterior part of his thigh on to the anterior abdominal wall. Since his injury he had been constipated, and had noticed increasing difficulty in micturition. On being more closely questioned, he admitted to lifelong difficulty in micturition when in the presence of others, but this was judged to have a psychological basis and to have no relation to his organic condition.

On examination of the patient, he was tender to pressure over the fifth lumbar spine. There was obvious wasting of all muscle groups in the lower limbs, including the glutei, but he could carry out, with decreased power, all movements except dorsiflexion of the ankles, in which there was frank foot drop on the right side. All tendon jerks were reduced to mere flickers on both sides, whilst the plantar responses were flexor in type. Hypoalgesia to pin-prick was demonstrable on the outer aspect of the calves in the fifth lumbar area, but there was no interference with deep sensibility. All abdominal reflexes were brisk, and no further abnormalities could be displayed at any higher level. X-ray films of the lumbar and sacral regions revealed defects in the neural arches of the third, fourth and fifth lumbar and first sacral vertebrae (Figure 1), with considerable expansion of the vertebral canal. There was an appearance very suggestive of a fracture of the fifth lumbar lamina. Lumbar puncture was attempted in the fourth to fifth intervertebral space, but no fluid could be obtained, so a further puncture was made in the second to third space, from which cheesy material was aspirated. The histological report on this matter was as follows: "Thick, sebaceous material in which objects resembling degenerate squame cells are seen."

At operation on October 13 a mid-line lumbar incision was made and the muscles were cleared from the bifid laminae. On incision into the *dura mater*, yellow fluid escaped, upward extension of the exposure being required. At the level of the third lumbar vertebra, sebaceous material and hair lay free within the cerebro-spinal fluid amongst the roots of the *cauda equina*. This material escaped from a capsule which was intimately adherent to the nerve roots. This capsule was opened, and all its contents were removed entirely. An attempt was then made to unravel the capsule from the nerves, and a great deal of it was painstakingly removed, but there was no certainty that it had been taken out in its entirety. Some injury to certain nerve roots was observed, but it was not judged to be extensive.

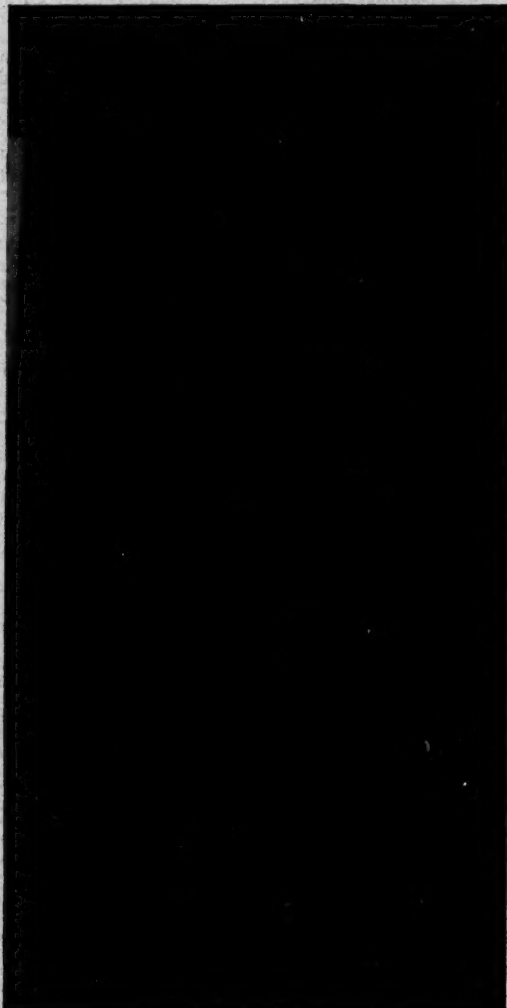


FIGURE 1.

X-ray film of patient B. *Spina bifida occulta* of third, fourth and fifth lumbar and first sacral laminae associated with appreciable widening of the vertebral canal in the same region. Note that the latter is distal to the site of the actual tumour.

On macroscopic examination, this could have been only a dermoid cyst. The full pathological report is as follows:

The oval cyst wall, stuffed with cotton wool, measured 4 x 3 cms. Microscopically the structure was typical of a dermoid. Flakes of partly keratinised cells cover the surface of the epithelium which is supported by a vascular, alveolar tissue containing a few bundles of

nerve fibres, and, in one field, what resembles glial tissue. The remainder of the specimen is quite a large amount of coagulated sebaceous material mixed with hairs.

The patient's post-operative course was slow but satisfactory. Immediately after operation all movements were still weak, and there was no movement at all in the toes of the right foot, which had been chiefly affected before operation. He was unable to pass urine, so tidal drainage was instituted. Within ten days this was discontinued, and he had no further urinary difficulties. Within five weeks he was able to stand in a brace and take a few steps with support. Two weeks later he could walk with crutches, and in another ten days he was back to his pre-operative state, with strong dorsiflexion of his left foot, and weak dorsiflexion on the right side. The fifth lumbar hypoaesthesia remained. Repeated attempts to communicate with this patient for follow-up purposes have been fruitless.

Pathology.

The basic embryology of the central nervous system provides a clue to the formation of these odd inclusion tumours. Reference to Figures II, III and IV reveals the formation of neural tube from neural groove, and the infiltration of mesoderm forming the supporting structures and vertebral column. It should be readily apparent that any failure in the accurate infolding of the groove could leave an epithelium-lined track down to the tube, which might persist as an actual sinus or become shut off

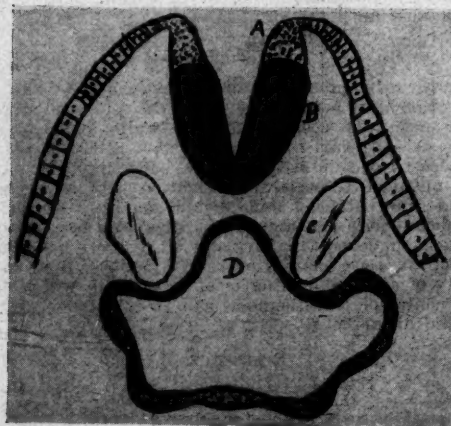


FIGURE II.

Infolding of surface epithelium to form neural fold. A, epithelium; B, neural fold; C, dorsal aorta; D, foregut.

from the surface, an epithelial inclusion being left in or adjacent to the adult spinal canal. Such tracks and cell rests are frequently combined with a failure of proper development of the associated supporting structures, the commonest of which is lack of fusion of the vertebral laminae, known as *spina bifida occulta*. A point of clinical interest is that the associated anomaly is frequently at a different level from that of the tumour itself. It is obvious that where there is an actual patent sinus leading from skin to vertebral canal, there is a very real path of infection to the meninges. In the event of an organism gaining entrance, meningitis is the logical result. Once the immediate acute episode has been overcome, organisms may lie dormant along the track, or within any associated dermoid, ready to flare up under favourable conditions into another acute attack. Thus, in cases of unexplained, recurrent spinal meningitis, any examination is incomplete without a search for a sinus along the patient's back. Any local pigmentation, dimples or hirsute patches must be especially examined, for these may be the fingerpost pointing to the external opening of such a sinus.

It is not amiss to mention here the sacro-coccygeal dermoid and sinus. This is a common complaint, and placed as it is distal to the lowest limits of the thecal sac, it does not communicate with the meninges or act as a source of meningitis.

Clinical Features.

The total number of cases reported, though increasing greatly in the last ten years, is still not great; but it is very striking to note the extremely wide range of ages of

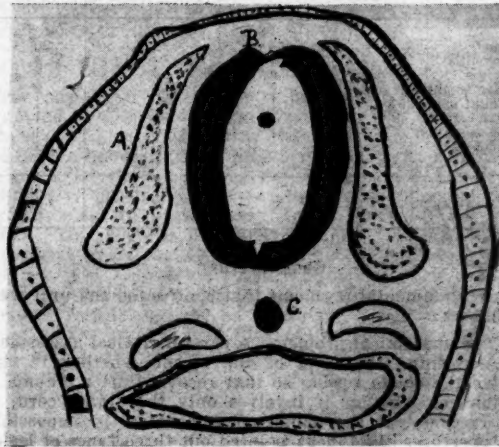


FIGURE III.

Developing neural tube. A, somite; B, neural tube; C, notochord.

those affected, children and adults, both young and old, being equally involved. List (1941), in a review of 56 cases, found that the average age of 22 patients with spinal

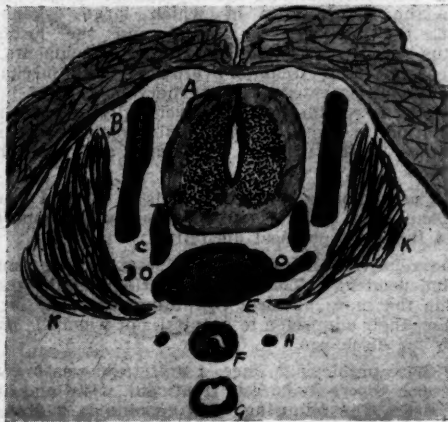


FIGURE IV.

Developing spinal cord and vertebral column. A, spinal cord; B, neural arch; C, posterior root ganglion; D, vertebral artery; E, centrum; F, oesophagus; G, trachea; H, sympathetic trunk; K, paraxial muscles.

epidermoids was 29.8 years, with an average duration of symptoms of 6.3 years. The equivalent figures for 16 dermoids were 28.3 and 9.6 years respectively. The actual signs are simply those of any slowly growing spinal tumour, and as in more than half the recorded cases the tumours occur below the level of the last thoracic vertebra, conus and cauda-equina type lesions prevail. Pain, both in

the back and in a sciatic distribution, is a common feature, and weakness of one or both legs appears later. Bladder and bowel dysfunction is frequent, whilst recurring meningitis is associated with a sinus opening upon the skin of the back. Objectively, spinal deformities, as scoliosis, are observed, and associated congenital anomalies may be found—for example, club foot. The tendon reflexes are increased when pressure is upon the spinal cord, producing a spastic weakness of the limbs, whereas a flaccid paresis with depressed jerks is seen customarily with cauda-equina involvement. Sensory changes may be mapped out in various dermatomes, whilst interference with the kinesthetic sensations of position and vibration appears, as a rule, at relatively later stages.

X-ray examination of the spine can be of considerable value. Allusion has already been made to *spina bifida occulta*; fusion of the vertebral bodies is another associated anomaly, whilst bifid spinous processes have also been reported. Widening of the vertebral canal has frequently been observed, the increased distance between the articular processes in antero-posterior views over several vertebrae making a most striking picture and indicating slow growth with prolonged pressure by the tumour (Figure I).

Lumbar puncture provides findings parallel to those in any other type of tumour—usually an increase in protein content of the cerebro-spinal fluid, which becomes extreme when a total blockage to the flow of fluid has developed. If perchance the needle is inserted into the dermoid itself, no fluid is obtained, unless it is cystic fluid from the tumour; but on occasions dermoid material may be withdrawn by suction, which makes the diagnosis absolute.

Myelography is of use as an adjunct to localization, and indeed is most necessary in cases in which no sensory level can be determined clinically to provide a guide to the tumour. A diagnosis of spinal dermoid may be entertained in the presence of a long history suggestive of a spinal tumour associated with congenital anomalies of the vertebrae themselves, or with any other congenital defect elsewhere in the body. The coexistence of a dermal sinus makes such a diagnosis virtually certain.

Treatment.

The treatment of these lesions is operative removal of the tumour together with the sinus, when one exists. Dermoids may lie extradurally, subdurally or in an intramedullary situation; the extradural dermoids are easiest to remove *in toto* and have the best prognosis; the subdural dermoids have an intermediate prognosis, and the intramedullary the worst. When the tumour lies amongst the roots of the *cauda equina*, the adherence of the capsule to the roots is the all-important factor. If there is no adhesion, total removal and cure are likely; but when the capsule is widely adherent, it may be quite impossible to remove it all without risk of severe damage to the cauda. In such cases, the contents of the tumour are removed with as much of the capsule as it is judged possible to remove without risking excessive damage to the nerves; but after this procedure tumours have recurred, and so have required repeated surgical intervention. As a rule the operation is carried out in one stage, though Craig and Mitchell (1931) advise a two-stage operation on children, carrying out laminectomy as a first stage and removal of the tumour one week later.

Summary.

1. A case of intraspinal dermoid, treated surgically, is reported.
2. Notes on the history and nomenclature of these tumours are proffered.
3. The clinical features and pathology are discussed, and special reference is made to the coexistence of dermal sinus, with which spinal meningitis may be associated.

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WHAT OF THE PARAPLEGIC?

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BETWEEN the two World Wars the paraplegic became relegated to convalescent hospitals to linger on, not in discomfort (as many are insensitive), but in misery until death claimed yet another victim.

The advent of the second World War brought the problem to notice more forcefully, and this time it seems as though it will not be allowed to slip again into the vale of despondency. Such is an easy course that results in unnecessary wastage of manpower and in the condemnation to total invalidism of those who otherwise could regain their rightful place in a community. During World War II a spinal injuries centre was set up at Stoke Mandeville in Berkshire. This was followed by others in the United Kingdom, at places where the need was greatest—at Cardiff, at Eastbourne and in the coalfields and industrial towns of the Midlands. The war over, these centres were partly absorbed by the Ministry of Pensions for it was clearly realized that—properly run—these centres could be a source of inspiration to those affected and, what is more important, an economical success.

In this paper it is intended to record the treatment, and some experiences gained in the management, of patients admitted to the Paraplegic Unit of the Royal Perth Hospital.

During the early months of 1954 it became apparent that such a unit was needed to care for the steadily increasing number of people stricken with paraplegia from a number of causes. As can be seen from Table I, some 37 persons have passed through the unit or are still under care. In 20 of these cases the paraplegia was the result of

acute trauma, and for these patients no planned rehabilitation could, at that time, be arranged, as their physical recovery was uncertain and in most cases improbable. With this problem in mind, a unit was created to segregate these patients, and a plan of management was drawn up to see that a civilian paraplegic could once again resume his place in the community.

TABLE I.

Cause of Paralysis.	Paraplegia.	Quadriplegia.	Total.
Trauma	18	2	20
Poliomyelitis	4	—	4
Carcinoma	4	—	4
Vascular condition	2	—	2
Congenital anomalies	1	—	1
Myelitis	1	—	1
Spastic condition	1	—	1
Arachnoiditis	2	—	* 2
Total	35	2	—

ORGANIZATION.

The fundamental problems that confronted the unit were as follows.

1. The anatomical problem. A careful clinical assessment will determine the level of the lesion, whether it may involve the *cauda equina* so that recovery after trauma is possible, or whether it involves only the spinal cord, so that recovery, if the damage is extensive, is impossible. Holdsworth *et alii* (1953) pointed out three types of lesion when referring to thoraco-lumbar fractures. However, these may be regarded as fundamental at any level in cord damage: (a) complete cord and root division; (b) complete root escape; (c) incomplete cord and root escape.

2. The pathological problem. It has to be determined whether the lesion is progressive, or whether its natural pathological history has reached its peak, only to subside leaving a trail of destruction.

3. The physiological and biochemical problems. These concern the determining of which large organs—for example, the bladder, the bowel and the locomotor apparatus—are deprived of their cranial attachments. By such deprivation a circulatory problem, both mechanical and functional, is caused. Some nervous control by the severed cord is retained and can eventually be used in the rehabilitation of each patient. Biochemically there is interference with nitrogen, calcium and phosphorus metabolism.

4. The psychological problem. This is often a complex and difficult one, but one which assumes an importance equal to the more physical disability.

5. The rehabilitation problem. These patients must be made independent. Thus, rehabilitation should proceed alongside the more physical retraining and must be considered on their admission to hospital, not when the patient is ready for discharge.

With these problems to face, a unit team was organized to give each aspect its correct treatment, when and how it was needed. The following were appointed: a director—a member of the honorary orthopaedic staff; an assistant director; a resident medical officer; two well-trained sisters interested in paraplegia; three physiotherapists; an occupational therapist; a social worker.

The unit also coopted the services of the honorary urologist, the senior honorary orthopaedist, an honorary neurosurgeon, an honorary plastic surgeon and an honorary physician.

The organization of the unit arranges that the patient presenting an acute problem is managed in one ward under the care of one staff. A routine management is decided upon, and this is commenced before the director sees the newly admitted patient. This step in organization is extremely important in order to make certain that no

initial mistakes are made. Such mistakes—for example, the passage of a large catheter in the ward by an inexperienced officer—can lead to at least delayed rehabilitation; it can even lead to bladder infection and pyelonephritis. The patient presenting a subacute and long-term problem (not chronic) is then managed in a specially equipped ward in the convalescent part of the Royal Perth Hospital at Shenton Park. Here the patients are taught to live with their disability and to overcome it. At the onset rehabilitation commences by the taking of a social history, and a plan is decided on for reemployment with the close cooperation of the local Commonwealth Rehabilitation Department.

MEDICAL MANAGEMENT.

The care of each patient as he passes from stage to stage is guided by a strict adherence to detail laid down by the honorary medical staff controlling treatment. It is fundamental for the care of the paraplegic that each stage must be carefully worked through. We have divided rather arbitrarily, but usefully, the care into the following three periods: (a) An acute phase, which lasts for some six weeks. This stage is prolonged at times by neurosurgical procedures or by the need for rest and posturing in poliomyelitis. (b) A subacute phase, in which physical rehabilitation is given pride of place, so that bowel, bladder and physical retraining can be developed. (c) A terminal phase, in which the activities of daily life are insisted upon, and during which occupational therapy and rehabilitation are fundamental.

The Acute Phase.

In the acute phase medical care can be considered under the following five headings: the aetiology of the disability; care of the bladder and bowel; care of the skin; care of the lower limbs; supportive therapy.

Aetiology.

The aetiology of the paraplegia may be obvious, and two large groups can be recognized—(i) the traumatic, and (ii) the non-traumatic. The latter is the smaller, but contains many patients for whom surgical treatment may be beneficial. Removal of the cord tumour and drainage of the paravertebral abscess may allow recovery. In the former, certain well-defined principles need attention. Operation and exploration in our opinion are justified only in the following circumstances: (i) in incomplete paraplegia which is growing worse; (ii) as a method of stabilizing the unstable spine to aid nursing; (iii) in all cases of cervical injury, with or after reduction of a dislocation, in which rapid decrease in the quadriplegia does not occur; (iv) in the rare case of acute paraplegia without fracture in which an extradural clot may be present; (v) when paraplegia has its onset some hours after an accident; (vi) in some *cauda equina* lesions without gross dislocation with a fractured lamina.

In a review of over 1000 patients treated at the Stoke Mandeville centre, Guttman (1954) holds that he does not believe that open operation has ever helped a paraplegic to recover. American opinion does not agree with this conservative British opinion. Laminectomy is performed frequently. Covall *et alii* (1953) state that only by laminectomy can an accurate assessment of the pathological state be obtained, that 2% to 3% of patients may be benefited and that the incidence of spasm is reduced. Psychologically, operation is good therapy as patients feel that all has been done for them.

The unstable fracture of the spine does need fixation. This may be attained by internal fixation or by the application of a plaster cast. We do not favour this latter method; such plaster casts fit when they are first made, but after a few days, during which time the patient wastes, they cause pressure ulcers of the sacrum and hips (see Figure 1). We support Holdsworth *et alii* (1953), who divide cases into "stable" and "unstable", and reserve operation and fixation for the latter.

Care of the Bladder and Bowel.

In the initial phase a small Foley catheter—inserted in the operating theatre, to prevent infection—is used to drain the bladder. The bladder is washed out with normal saline. Intermittent distension with continuous washout between is resorted to in order to maintain a normal bladder capacity. Paraplegics have hypercalcaemia and hyperphosphaturia due to the upset in mineral balance and alteration in circulation, occasioned by the denervation of the lower extremities. Phosphatic deposits and bladder infection are the real problems of bladder management. The following four measures are advised to combat these: (a) the maintenance of acid urine; (b) the administration of "Amphogel" to decrease calcium intake; (c) the arranging of a diet of low calcium content; this has to be considered in relation to protein intake, and usually a moderate calcium quota is allowed; (d) careful attention to adequate drainage; early mobilization is desirable, as with mobilization the abnormal biochemical state ceases and urinary drainage is improved.

The bowel is controlled by the use of enemata on alternate days at a fixed time to start a conditioned reflex.

Care of the Skin.

Immediately after injury or the onset of the pathological process, the paralysed parts need special care until automatic functions recover and retraining can be commenced. The major immediate problem is that of the skin. Human skin is an organ capable of respiration, secretion, excretion and protection. Of these, protection must be encouraged. Without waiting for specific instructions, and indeed in the absence of them, careful skin toilet and regular two-hourly positioning (with the avoidance of all twisting of the spine at the fracture site) will go far in preventing major skin catastrophes. To a paraplegic a skin ulcer is a major problem. Pressure areas appear quickly (see Figure 1).



FIGURE 1.

Pressure sores, illustrating those caused by immobilization in a plaster cast.

The paralysed state, associated with vascular stasis, causes anoxia and with pressure, ulceration. The early care of the paraplegic is largely nursing care, as for only a few can operation be very helpful. The prevention of pressure areas—the curse of a paraplegic's life—can be achieved only by the development of a sense of responsibility in every nurse engaged in the care of such patients. Attention to detail is essential; the reporting and protection of the slightest area of reddening of the skin will be repaid by the deep gratitude of the patient. Two-hourly positioning has the following three important functions: (i) it prevents venous stasis, thus enhancing circulation and venous return; (ii) it is necessary to stimulate that most important protective function of the skin; (iii) it helps to prevent urinary stasis.

In the early stage many patients are acutely ill, needing blood transfusion, and gastric aspiration to prevent dis-

tension, so that nursing is often tedious and arduous. A kind word at the right time, a question answered correctly and in a manner easily understood, will be appreciated. Attention to every minute detail relating to bed clothes, pillows and mattresses is absolutely essential.

Care of the Lower Limbs.

Paralysed and inert, the lower limbs must be positioned correctly in each change of posture. The physiotherapist's aim is to keep oedema down, keep the joints mobile and prevent contractures (see Figure II).



FIGURE II.

Lateral nursing position. Notice the position of the lower limbs, with a pillow between the knees and ankles. The lower foot in the acute stage is kept dorsiflexed by a pillow at the end of the bed; the upper foot can be kept dorsiflexed by a soft pillow or sandbag.

Paraplegic patients need particular care, as they usually present the following features: (i) muscle wasting; (ii) a disinclination to eat, and abdominal discomfort; (iii) progressive anaemia due to a number of causes, for example: (a) initial trauma and the protein lost subsequent to it at and around the fracture site; (b) the increase in the circulating volume of blood. The lower extremities, being paralysed, have a much larger capacity for fluid than in the normal subject, and thus blood volume is increased with hemodilution. Compensation is arranged by the hematopoietic centres. To combat such a natural history the following investigations must be commenced on the patient's arrival in the ward: (a) The hemoglobin value must be estimated two or three times a week until it becomes stabilized. (b) The blood urea level must be watched as a check on nitrogen metabolism. (c) The serum protein content must be estimated.

Treatment.

It is essential that in the early stages these patients receive blood transfusions as they require them. A hemoglobin value of 95% to 100% must be aimed at if the well-being of the patients is to be maintained at such a level that they are able to cope with their dietetic and urinary problems. Such correction of anaemia helps to prevent urinary complications.

It is essential that such patients receive a protein-rich diet to prevent muscle wasting, which is prevalent in the first three or four weeks of management. Such protein intake is difficult to arrange, and needs the careful attention of a dietitian who insists that the patient takes this food regularly and at frequent intervals.

Drug therapy, including the administration of testosterone, 15 milligrammes per day, also helps to prevent muscle wasting.

In the complicated case, when the patient is brought to a unit with bed sores, infected urine and joint contracture, many months of attention by the expert medical team may

be needed to control these problems before the patient's rehabilitation commences in relation to work and the future. Teamwork is absolutely essential. Every case needs thorough discussion.

The Subacute Phase.

The subacute stage may start as early as the week after injury, or as long as six weeks after, depending on the original severity of the injury and the rapidity of recovery. Before this stage can commence, we consider it essential that the medical officer shall visit the patient with the important task of discussing frankly and thoroughly his or her problem. At such a discussion the question of prognosis should be broached as fully as possible. The physiotherapist now becomes a more important member of the therapeutic team. It is his or her job to train the muscles thoroughly, to prevent joint contracture and, finally, to assist the patient to take his or her first steps. Just how long this retraining takes depends on the following factors: (a) the patient; (b) how quickly the patient progresses in his physical rehabilitation; (c) rehabilitation in relation to toilet habits (at times a long and painstaking process); and finally (d) the patient's mental outlook.

The patients must be instructed carefully and fully in each step, no question remaining unanswered. All the time a careful medical check-up is needed week by week into their dietary requirements, their circulation and kidney function, their blood pressure. From time to time certain specific treatment—for example, blood transfusion, small operations on the bowel and bladder—may be needed. This stage is guided by the following well-defined principles: (a) Emphasis is placed on the development of the upper limbs. (b) Great importance is laid on gradual regaining of balance—first sitting, then with the aid of calipers and crutches. (c) Finally attention is given to three-point and four-point walking. (d) A regular toilet habit must be inculcated—the establishing of a conditioned reflex. We have had special toilets and bathrooms arranged so that this retraining can go along unimpeded. (e) Psychological stimulation is constantly required.

Each one of these points needs special care. Development of the upper limbs must be commenced as soon as possible after the patient has been admitted to hospital, by weight-lifting exercises, first in bed and then in the gymnasium. The regaining of balance is difficult and takes a long time. The patients must be taught to use visual and aural senses in helping to maintain the erect position. A regular toilet habit is essential to the social success of the treatment of paraplegics. The simple experiments of Pavlov are explained to each patient in turn, and he is encouraged to develop conditioned reflexes if this is possible. After careful consideration of the type of bladder that can be expected from the neurological lesion, most patients, after a long period of time, will develop a satisfactory toilet habit provided that they are willing to undertake the arduous, and at times very irksome, repetitive therapy that is necessary. The matter of oral intake of fluid must be carefully controlled so that the patients will remain socially adequate during twenty-four hours, and particularly in the evening when they are socially engaged.

Psychological help is of a general type. Usually the stimulant of being one of a number of patients is enough to help the patient to overcome psychological depression. However, at times, and principally when the patient has been inadequately managed prior to being taken over into special care, it may be necessary to have advice and help from the psychiatrist.

The Terminal Phase.

With the patient reaching a stage of being out of bed in his own mobile chair, in control of his natural functions, then recreation is provided, and finally retraining. Such rehabilitation is planned thoroughly so that when each patient reaches the stage of requiring an active occupation the work is awaiting him. Sports must be encouraged. Indeed, a competitive spirit is to be fostered, for the following sports can be engaged in: basketball, archery, billiards, ping-pong, javelin throwing, darts, fencing, skittles,

badminton, climbing ropes, swimming and polo. It is well known that paraplegic teams can compete very favourably indeed with fit persons trained to use wheelchairs for these sports. Overseas, annual Paraplegic Olympic Games are held at Stoke Mandeville with teams from Europe, America and Canada competing—an incentive for Australia eventually to send representatives.



FIGURE III.
Archery practice.

Rehabilitation is of fundamental importance to patients. It is necessary to start such rehabilitation soon after their admission to the unit so that the habit of occupation is not lost. In the field of restoration to work, patients can be retrained in carpentry, joinery, cabinet-making, cobbling, shoe-making, splint-making, watch-making, painting and a host of clerical occupations. The housewife can once more



FIGURE IV.
Javelin throwing.

look after her family, the husband become once more the income earner. Already a start in rehabilitation is being made in the precincts of this unit; one patient has become interested in net-making in relationship to the fishing industry, thus acquiring an occupation which he can carry on after having left the unit. A second patient is becoming an efficient boot-repairer, and a third will ultimately follow suit.

By the active cooperation of the Commonwealth Rehabilitation Department, in those cases eligible for their assistance, each patient is discussed and his rehabilitation is planned, so that after leaving hospital he is not thrown back on to the invalid pension for the rest of his life.

EXPERIENCE GAINED IN THE UNIT.

Constant care must be given to the minutest detail, as is shown by the following case:

Mrs. S., aged fifty years, had quadriplegia due to a dislocation of the sixth on the seventh cervical vertebra. She had a very stormy passage, but her skin remained intact. Then, on account of pyrexia due to urinary infection, she had to be transferred back to the "acute" ward. She was in transit and on the X-ray table for one and a half hours only. On her arrival in the ward there were early signs of a large pressure sore; this progressed and is still under care. Had more care been exercised during transit, this would have been prevented.

In the management of the bladder, urinary infection must be guarded against all the time. Unless all measures—the use of acidifying agents, care in diet, and, above all, aseptic methods of catheterization—are strictly adhered to, infection will inevitably occur. We have not yet overcome this problem, as is shown by the following case:

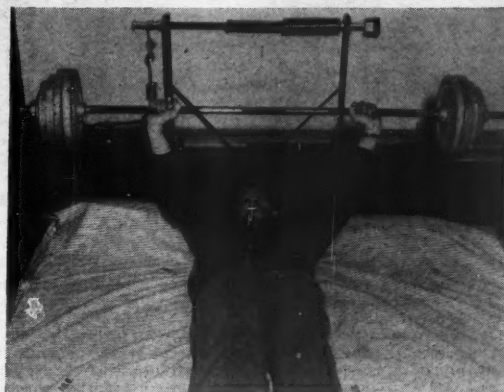


FIGURE V.
Weight lifting—150 pounds.

K. had complete paraplegia due to a fracture-dislocation of the fourth on the fifth thoracic vertebra after a motor-car accident. He was admitted to hospital and had a urethral catheter passed. This traumatized the ureter and it was necessary for the urologist to carry out a suprapubic cystotomy. The patient's bladder became infected, and the infection was with difficulty controlled. The control was not successful until the patient had been mobilized, his suprapubic catheter had been removed, anemia had been corrected, his fluid intake had become adequate and a course of antibiotics had been used.

All problems pertaining to paraplegics must receive concomitant care—this is particularly so in the management of the bladder. This patient now has a reflex three-hour bladder and sterile urine.

A review of our traumatic cases does not permit more than a few conclusions to be made, as they have not yet been under review for a long period. The following conclusions are permissible: (i) The vulnerable areas are (a) the lower cervical area, (b) the upper thoracic area, (c) the lumbo-dorsal junction. This is well documented in the series reviewed by Holdsworth *et alii* (1953). (ii) The more cord left below the lesion, the longer the course of rehabilitation needed, and the greater the possibility of abdominal and flexor spasms.

The adequacy of retraining of bowels and bladder is shown by the following two cases:

S., a girl, aged eighteen years, had paraplegia due to a fracture-dislocation of the eleventh on the twelfth thoracic vertebra. She was finally discharged from the unit after nine months of therapy with a perfect three-hour reflex bladder and a perfect automatic bowel which functioned once a day, and she was socially adequate in all ways.

G., another girl, aged seventeen years, had paraplegia due to a fracture-dislocation of the fourth on the fifth thoracic

vertebra. After eleven months of treatment in hospital she had good bowel function once a day and was rapidly developing a good reflex bladder.

We are certain that if retraining is adequate then very few patients need have bowel and bladder function that will be "socially inadequate".

Suprapubic cystostomy has little place in the permanent care of the paraplegic, as is illustrated by the following two cases:

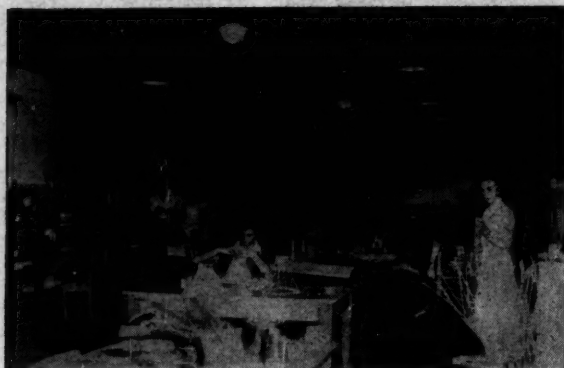


FIGURE VIIA.
Occupational therapy.

G., a man, aged twenty years, sustained a fracture-dislocation of the seventh on the eighth cervical vertebra with quadriplegia which did not recover. He was admitted to the unit eighteen months after the injury with sacral pressure areas, gross myasthenia, wasting and a suprapubic cystostomy. It was proved by investigation that urethral micturition was possible; but some nine months were to elapse before the suprapubic cystostomy could be closed, as this man had gross urinary infection, a bladder stone and stricture of the prostatic urethra. All these could have been prevented by the initial use of urethral drainage.

B., a man, had a fracture-dislocation of the sixth on the seventh cervical vertebra. Urethral drainage proved difficult, and it was thought that a suprapubic cystostomy might help bladder drainage. This was established, but only made further complications in an otherwise straightforward case, for the suprapubic tube quietly caused a vesico-colic fistula, and this suprapubic cystostomy has only just been closed. This procedure, we now feel certain, was a mistake. To sum up, therefore, it can be stated that suprapubic cystostomy, as a permanent method of bladder drainage, only enhances infection, allows stones to form, and permits the bladder to contract to such an extent that no amount of dilatation later will distend it to a useful size.

The group of poliomyelitic patients we had under review were all severely paralysed. One became an incomplete paraplegic—it was an interesting observation backed by Sharrard's (1955) recent work that no patient with a muscle power at "0" would regain useful power in that group.

One patient with secondary carcinoma had six weeks' intensive treatment which allowed her to be discharged from the unit in a wheel-chair. In the other five such patients, despite our efforts, the condition gradually progressed; but adequate nursing did make them more comfortable. The miscellaneous group included two with vascular anomalies, one with transverse myelitis, a spastic, and one with amyotrophic lateral sclerosis.

No patient in the acute stage admitted to hospital after the unit was formed developed pressure areas except in the case quoted above, but we inherited five patients who had severe pressure sores. Now in all save one the sores are healed. This patient, a paraplegic (now into the second year since his accident), awaits closure of the sores by a plastic procedure, his problem being also psychiatric.

The optimum period for a patient to be in hospital varies with the lesion and the patient, and cannot be stated early after admission to hospital. From present indications, nine to ten months is about the minimum period during which a patient can expect to be hospitalized.

DISCUSSION.

The first year of the Western Australian unit has shown the possibilities of rehabilitation of a group of patients for whom to date nothing collective has been attempted in this continent. Individual efforts throughout Australia have been reported; but such disabled persons need the organized care possible only in a unit. They need to be given encouragement, and finally they need to be treated normally by their parents, friends and acquaintances.

On leaving the unit they should need no help at all in the daily problems of living. Anything short of this must be regarded as a failure. Even after their discharge from hospital, follow-up examination must be well planned to prevent any complications—for example, urinary infection, contractures, crutch palsies, small pressure areas. The medical problems of paraplegia are all interlocked; of these, urinary infection is the greatest, and can be prevented and treated only by the greatest vigilance in every detail. Munro (1953, 1954) adopted a tidal drainage routine, Guttman (1954) intermittent catheterization. We have not yet been able to give the former a trial. Bors (1951) gives a useful description of the type of bladder control that can be expected. This problem warrants much further study and attention.

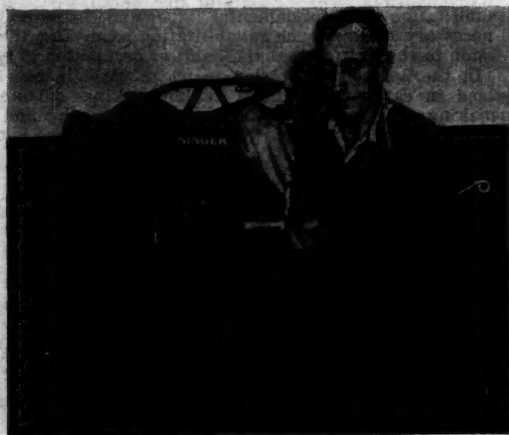


FIGURE VIIB.
Occupational therapy.

It is, unfortunately, true that the majority of people who have had efficient management for paraplegia at present have been ex-servicemen; the paraplegic centres in Great Britain were largely opened to cope with battle casualties. Now they cope with civilians under the Ministries of Pensions and Health. In the United States of America the Liberty Mutual Insurance Company estimated that by the establishment of an efficient service they had a return of 600% on their initial output for rehabilitation and medical management when compared with the previous cost of treatment by a haphazard method. With such a service much public money would be saved and unnecessary morbidity and death prevented. It would fill a big gap in our modern community. Australia has had no such self-sufficient units well established and functioning. Paraplegics find great difficulty in becoming completely rehabilitated. It is a slow process. They must compete with other injured workers. Since their capacity to become rehabilitated is slow, and because until now they have been regarded as "permanent invalids", they compete with handicaps. It is necessary for special arrangements to be

made. A further problem that confronts them is their reemployment in industry. Great Britain has regulations enforcing the employment of disabled persons in large factories on a quota basis. In Australia such employment depends on individual employers; in Perth we have the cooperation of a number. Until legislation is passed and until employers realize that these persons can be employed satisfactorily, our present problems will remain. The establishment of this unit has shown that these patients can be satisfactorily rehabilitated.

In conclusion, I should like to stress the problems of the Australian paraplegic. Units in this country can never be as large as those overseas. Nevertheless, there is a great need for such units to be inaugurated, so that by segregation and adequate team management these persons can be adequately managed.

SUMMARY.

This paper has attempted to emphasize the integration of the care needed in paraplegia. The individual aspects need very careful attention to detail and will be considered at more length in other papers.

ACKNOWLEDGEMENTS.

I wish to acknowledge the assistance I have received in this work from Dr. A. L. Dawkins, Senior Orthopaedist at the Royal Perth Hospital; from the Board of Management of the Royal Perth Hospital; and from the Commonwealth Rehabilitation Department in Western Australia.

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THE DOCTOR AND MARRIAGE GUIDANCE.

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THE high incidence of divorce and domestic discord is one of the disturbing features of present-day society in most, if not all, of the civilized countries of the world. In Australia, between 7000 and 8000 marriage partnerships are terminated in each year by divorce, many others are broken through the separation of the partners by desertion or by mutual consent, and in many more homes there is such a degree of permanent discord that there is no longer any semblance of partnership between husband and wife.

It is now universally recognized that this widespread disruption of home and family life is something of great public as well as private concern; but it is not as widely recognized that it is an inescapable factor in personal, social and national health.

Every practising doctor finds many illnesses in men, women and children which appear to be precipitated, intensified or prolonged by persistent domestic strains—

notably the psychoneurotic illnesses and many of the so-called stress diseases. It is becoming generally accepted that one of the most influential of the known and controllable factors in the causation and persistence of mental illness is the deprivation of love and security, particularly in early childhood, in the home. This appears also to be a strong factor in the causation of such social disorders as delinquency, vandalism and crime, and of much of the ill-defined emotional instability which brings destructive social, political and industrial discord.

The cost of all these disorders to the community is incalculable. Apart from the heavy and widespread burden of misery, bewilderment and despair borne by those who suffer from them, and by their relatives and friends, there is the fabulous cost in public and private money, of hospitals, institutions, police courts and gaols, and of devoted man-power and "woman-power". There is also an incalculable hidden cost by virtue of the fact that most of the sufferers are consumers but not producers, and a far-reaching cost in the passing on of negative or destructive psycho-pathological influences to future generations.

At the beginning the community set out to deal with severe mental illness and crime simply by segregation of the sufferers and criminals in asylums and penal establishments respectively. As time went on, some attempts were made to treat the mentally ill and to reform the criminal, with partial success in each case. These efforts led to the recognition of the need for earlier diagnosis and treatment, and still more of prevention; and mental hygiene clinics, youth clubs and other social institutions came into being. Special children's courts, helped by probation officers and other humanitarian people, have carried out increasingly valuable work.

But no coordinated large-scale attempt has yet been made in Australia to deal with these personally and socially destructive abnormalities at the point of their most frequent onset, the home and family. It would appear that such a nation-wide approach would make possible one of the most influential and far-reaching programmes of preventive medicine and preventive psychiatry ever undertaken by the community, one which would bring together and greatly extend all the fragmentary and insufficiently coordinated activities in existence for that purpose. By its very nature any such programme would require expert medical leadership and support.

Some very important moves in this direction have been made during recent years in many countries of the world. Many existing social welfare organizations have directed increasing attention to the welfare of the family, and many new family welfare organizations and marriage guidance councils have come into being. There has also been a rapid growth in the international coordination of the many efforts to promote more stable home and family life. At the World Congress on Family and Population in Paris in 1947, which brought together representatives of 27 nations, the International Union of Family Organizations was formed to coordinate and develop the many and diverse approaches to the problem. Its final constitution was adopted by its First General Assembly in Geneva in 1948, during which year it was granted consultative status by the Economic and Social Council of the United Nations (U.N.E.S.C.O.) under Category B, and recognized by that organization as a consultative body. From its headquarters in Paris the International Union of Family Organizations has grown steadily year by year and has carried out many important investigations which have been and will be of great benefit to all who seek to promote better family life.

It is natural that the orientation of family welfare services will vary to some extent in different countries. For example, the main emphasis in Europe has been in the direction of the external factors in family welfare, such as economic stability, housing, nutrition and clothing, with little or no attention to the inner dynamics of the marriage relationship as such, or to the other domestic relationships. The importance of these central aspects of family welfare was clearly recognized; but the marriage relationship was

thought to be too private and personal to be made the subject of any community programme for family welfare.

Experience in America, Britain, and more recently in Australia and New Zealand, where the main external factors are accepted to some extent as government responsibilities, is that no such external assistance as can be provided by the welfare State can be fully effective unless the marriage relationship, on which the whole family life revolves, is or becomes healthy and stable. It has also been abundantly proved in these countries that adequate personal help can be given through what is called marriage counselling, and that this help is welcomed by an increasing number of married couples who find difficulty in straightening out their own misunderstandings and conflicts.

This counselling work has also made it clear that there is an even greater need for universal first-class education and preparation of young people for the complex and far-reaching responsibilities of marriage and parenthood far beyond the many disconnected and fragmentary attempts which have so far been made.

A full account of this Anglo-American experience was given at a conference of the International Union of Family Organizations (I.U.F.O.) at Oxford in 1953, which resulted in the formation (at the Lisbon conference in 1954) of the Commission on Marriage Guidance of I.U.F.O., as a permanent body reporting periodically to the General Council of I.U.F.O. The term "marriage guidance" had originated in England.

From all the diverse channels through which stable marriage relationships could be promoted, the operative field of marriage guidance has been deliberately confined to include the provision of three specific types of service—general preparation for marriage, specific preparation for marriage, and marriage counselling and conciliation. The general preparation for marriage seeks to help and guide young people at each stage, from early childhood to the point at which they enter into relationships with the opposite sex with the expectation that they will lead to marriage. The specific preparation seeks to help and guide them through courtship and the engagement period, and to prepare them for marital adjustment in many areas—physical, emotional, spiritual, economic and social—as well as to give them instruction in the great arts of home-making and parenthood.

Marriage counselling is a form of brief "non-directive" psychotherapy, in which the whole atmosphere is permissive. It is an attempt to assist each partner to gain clearer insight into his own and his partner's feelings and attitudes, so that they are in a better position to decide their own future. There are, however, many married couples who are in need of help, but are unwilling to seek marriage counselling. In some situations of this kind a divorce court or some other agency may see fit to take the social responsibility of bringing some pressure to bear on the partners to seek some kind of reconciliation, and such help as may be offered under these conditions is termed marriage conciliation to distinguish it from counselling.

In Australia, as in many other countries, an increasing number of almoners, social workers and members of many social welfare organizations are doing valuable work in the promotion of family welfare. Doctors, teachers, clergy, probation officers and many other people are also giving valuable help in individual cases met with in their daily work. In Sydney and Melbourne some religious denominations have organized educational and counselling services within their own fields.

In addition to these self-limited organizations, widely representative community organizations known as Marriage Guidance Councils exist in each of the Australian States, based on the capital city, and affiliated councils are being formed in many large provincial cities and towns. The work of all the State Marriage Guidance Councils is linked up and coordinated by the National Marriage Guidance Council of Australia, and there is close affiliation with the National Marriage Guidance Council of Great Britain and with the International Union of Family Organizations in Paris.

This work in Australia is still in its infancy, although it is growing rapidly. At this stage in its progress it is greatly hampered by lack of competent man-power, and insufficient financial and other resources.

The educational work is being carried out on a broad front mainly through "Home and Family Weeks", conducted by request in many parts of Australia. On a more detailed scale it is being carried out by the various State councils alone and in cooperation with many other organizations through courses of lectures and the showing of suitable films, and also through private premarital discussions with engaged couples. There is need for a vast expansion of all of this work.

The counselling work is of two types. For people whose conflicts are thought to be relatively simple and straightforward, the honorary services of a small number of carefully selected and partially trained counsellors are available in each State. Those whose difficulties appear or are found to be deeper are generally referred to their family doctor or, if they wish it, to an appropriate specialist—gynaecologist, psychiatrist, paediatrician or physician—on an ordinary professional basis.

Throughout Australia the State Marriage Guidance Councils are trying to cope with the increasing demands on their services with a grossly inadequate number of counsellors. The standard of training of some of the counsellors is not yet regarded as satisfactory; but it is considered better to do the work in this way than to reject the many grievously troubled people who seek help and know of nowhere else to go for it. This view is supported and encouraged by the results obtained.

No adequate programme of marriage guidance can be carried out without considerable government assistance. This whole problem was carefully investigated in England by a "Committee on Procedure in Matrimonial Causes", under Mr. Justice Denning, and their report was presented by the Lord High Chancellor to the British Parliament by command of His Majesty in February, 1947. Among the conclusions in the report are the following:

The reconciliation of estranged parties to marriages is of the utmost importance to the State as well as to the parties and their children. It is indeed so important that the State itself should do all it can to assist reconciliation.

There should be a Marriage Welfare Service to afford help and guidance both in preparation for marriage and also in difficulties after marriage. It should be sponsored by the State but should not be a State institution. It should evolve gradually from the existing services and societies just as the probation system evolved from the Court Missionaries and the Child Guidance Service from the children's clinics. It should not be combined with the judicial procedure for divorce but should function quite separately from it.

It should be recognized as a function of the State to give every encouragement and, where appropriate, financial assistance to marriage guidance as a form of Social Service.

Since the presentation of this report the work of Marriage Guidance Councils in Great Britain, and later in Australia, has been subsidized to a varying degree by the respective governments.

Even with the necessary financial help from State governments, it is not yet clear in Australia whether enough suitable people will be found who can give the necessary time for training and for teaching and counselling as honorary services. It may be that as the work expands further, as seems inevitable, a new profession will come into being to carry it out alongside the State-sponsored programmes. Such professional marriage counsellors may come from the ranks of social workers, teachers and clergy and other trained people who will undertake the necessary training and supervision. It offers a most creative field of service to many different organizations and professions.

What part may the doctor have in this work? In his daily work of diagnosis and treatment of disease he is constantly brought into close contact with domestic discord,

which is frequently an important factor in the causation, the severity, and the persistence of the trouble. If he has undertaken some serious study of the psychodynamics of domestic relationships he is in a unique position to recognize and assess the disturbance, and to offer help, often at an early stage in the trouble, long before the partners might seek marriage counselling on their own initiative. His relationship with all members of the family may well give him a particularly good opportunity to use his knowledge and experience to the best possible effect.

In his important responsibility regarding the prevention of illness and the promotion of positive health, he may also use his special knowledge and his personal relationships with families to exert a far-reaching influence for good. He is honoured by the privilege of being admitted to the "inner circle" of many families as a trusted counsellor and friend, and he can learn much about the principles of human relationships and offer the right word at the right time in many potentially harmful situations.

In his wider responsibility as a citizen the doctor can sometimes give most effective leadership and help in such organizations as marriage guidance councils, and this will do much to keep this delicate educational and counselling work on sound foundations.

In conclusion, therefore, it is suggested that the special knowledge, experience and professional wisdom of the doctor, and his unique relationships with his patients and their families, can be of the greatest value in the rapidly developing and expanding work of marriage guidance—a social service which may well prove to be one of the greatest "combined operations" of the latter half of the twentieth century.

Reviews.

A Primer of Freudian Psychology. By Calvin S. Hall; 1956. London: George Allen and Unwin, Limited. 5½" x 8½", pp. 149. Price: 13s. 6d.

This book forms another centenary tribute to Freud in that the basic value of his contributions to normal psychology is emphasized by its author, Professor Calvin S. Hall. He writes in the preface: "One is dismayed by the prevalence of criticism based on incomplete understanding. Freud seems to have suffered more in this respect than any other major thinker of our times. His theories have been so widely misinterpreted and distorted that it is almost impossible for the unsuspecting reader to separate fact from fiction. . . . I have tried to avoid this mistake by keeping the possibility of making it in the forefront of my mind." Professor Hall has largely succeeded in summarizing Freud's essential views in a remarkably clear exposition of just under 140 pages.

The first chapter is a vivid assessment of Freud's scientific heritage and achievements, about which Hall writes: "What then was Freud? Physician, psychiatrist, psychoanalyst, psychologist, philosopher, and critic—these were his vocations. . . . One may prefer to think of him, as I do, as one of the few men in history who possessed a universal mind."

A convenient feature of this valuable book is that at the end of all but the first chapter Professor Hall provides the list of Freud's books summarized in each, together with the relevant pages.

Biochemistry of the Eye. By Antoinette Pirie, M.A., Ph.D., and Ruth van Heyningen, M.A., D.Phil.; 1956. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 320, with illustrations. Price: 35s.

DURING the past ten years great advances have been made in our understanding of the metabolic processes which take place in living cells, the correlations of the different enzyme systems and the sites of activity, but the picture is by no means clear yet for a single cell, let alone for a whole organ. In writing a book on the "Biochemistry of the Eye", A. Pirie and R. van Heyningen have attempted something which at the present time seems an almost impossible task. One of the reasons given in the preface for writing the book is that "the clinical study of ophthalmology can be made more interesting and fruitful if the biochemical processes of the normal and abnormal eye are understood". Everyone will

agree with this, but the authors continue: "Such a real basis of knowledge does not yet exist, but perhaps the fragments presented here may show up possibilities of investigation and treatment in clinical work and encourage the use of biochemical techniques." There are 310 pages of text in the book, so a good deal of ground is covered in ten chapters. There are 32 pages on the composition of the lens and 55 pages on its metabolism with another 50 pages on cataract.

Again, in the preface the authors state that "many biochemists will be irritated by the scrappiness of the biochemistry, just as clinicians will be dismayed by the treatment given to clinical ophthalmology". In the section on the metabolism of the lens the greater part is taken by the elementary biochemistry of the cell and attempts to apply this to the lens. To the biochemist this is of little value and the clinician will be hard put to understand its relation to the conditions of the eye.

The chemical aspects of vision are treated at great length, but here again the application is not obvious.

The cornea, retina, vitreous body and aqueous humour are also considered, and there is finally a good section on ocular effects of nutritional diseases. There is a great deal of interesting material in the book, but it has to be dug out. As a book of reference the book could be very useful to the research biochemist. It is doubtful whether the clinician will get much from it to assist him in his practice.

Sick Children: Diagnosis and Treatment. By Donald Paterson, M.D. (Edin.), F.R.C.P. (Lond.), D.P.H. (Eng.), with the assistance of F. S. W. Brimblecombe, M.D. (Lond.), M.R.C.P. (Lond.), D.C.H.; Seventh Edition, 1956. London: Cassell and Company. 8½" x 6", pp. 603, with illustrations. Price: £2 2s.

REGINALD LIGHTWOOD's recent revision of Donald Paterson's little book called "Sick Children" has brought it back to the place that it formerly held as one of the very best, perhaps the best, of the small manuals on diseases of childhood. If one accepts the limitations that must be found in a book that surveys the whole field of children's diseases in 500 pages, then it is hard to criticize this book. It is practical, up to date, clearly written and well produced. The student is not likely to find any better text-book on this subject. The practitioner who wants a book that gives him a sufficient discussion of the important subject of paediatrics and some brief reference to the less important ones will be admirably suited too. A great deal of the old book has been radically revised. The chapters on history taking and clinical examination, the care of the premature infant, diseases of the heart and circulation, and on rheumatism have been almost entirely rewritten. A much more adequate survey of diseases of the nervous system, of tropical diseases, of diseases of the heart and circulation, and of rheumatism is included. The general practitioner will find great value in some of the appendices, particularly the one on drugs and their dosages. The other appendices deal with vitamin preparations in common use, electrolytes of the body fluids and ossification data.

We are very glad to welcome and recommend this little manual, particularly for the part that it is going to play in student teaching.

Emotional Problems of Early Childhood. Edited by Gerald Caplan, M.D.; 1955. London: Tavistock Publications, Limited. 9½" x 6½", pp. 558. Price: £2 2s.

It is timely and salutary to find a book which brings a critical appraisal of current thinking and practice in the field of child psychiatry. This volume, edited by Dr. Gerald Caplan, offers a representative coverage of papers presented at the International Institute of Child Psychiatry in Toronto during 1954 by psychiatrists, psychologists and social workers.

Concentration upon the child as an individual has been extended to include interpretation and therapy which is family-centred, with reference to the sociological values of community cultural patterns. Thus the child is considered in his total functioning as he attempts to adjust himself to an increasingly complicated social milieu.

Of special and challenging interest is the research into the socially isolated child, described variously as "autistic", "atypical" and "symbiotic-psychotic". Although they have presenting symptoms in common, these children, recognizable by their variants in early years, permit of contrasting opinions as to the origin of their social immaturity. All observers, however, are agreed that they present the earliest recognizable forms of psychoses in childhood. While explanations range from hereditary predispositions and birth

traumata to purely environmental pressures, there is a general belief that a defective mother-child relationship is primarily responsible for the perpetuation of personality disorders in a child.

This theme leads naturally to consideration of the effects of interference in an interpersonal relationship by temporary or permanent separation, of which hospitalization and institutionalization respectively are examples. Contributors realize that further research is needed into the degree of vulnerability, the tolerance level, and the capacity for adaptation and recovery of individual children.

Consideration of psychosomatic disorders, with their physical and emotional components, marks a further advance in interpretation of the behaviour of a child as a total whole. Not quite so convincing are the details of individual therapy, unless one realizes that verbal descriptions often fail to convey those intangibles inseparable from successful therapy.

Progress in Clinical Psychology. Edited by Daniel Brower, Ph.D., and Lawrence E. Abt, Ph.D.; Volume II, 1956. New York and London: Grune and Stratton. 9" x 6", pp. 462. Price: \$7.75.

In this book on clinical psychology, twenty-five contributors review the literature of 1952-1954. While the original Rorschach ink-blot test is still in use, there have been evolved many techniques of application and modifications in interpretation so that it is becoming more difficult to correlate the results of different workers. Dr. Piotrowski, who reviews the Rorschach method, states that over a hundred papers and books appear yearly on this subject, but he gives only fourteen references in his chapter. There is less optimism than previously over the value of thematic apperception tests, but the study of fantasy prompted by these tests may afford useful clues to motivation. More attention is being devoted to ego regression and retardation in children. We are still very far from a full understanding of what really brings about a change in a patient undergoing psychotherapy. Varieties of group therapy, such as play, art, music, books and drama, are discussed by G. Bach. In reeducation of aphasics stimulation, facilitation and motivation all play a part. In the chapter on the neurophysiology of higher processes there is a survey of recent experiments on animals, the results of which can have only a limited application to man. We are reminded of Hughlings Jackson's warning against confusing the localization of a symptom with the localization of a normal function.

This work is intended primarily for clinical psychologists, but psychiatrists will appreciate better what psychologists have to offer in the way of mental testing and treatment.

Campbell's Operative Orthopaedics. Editor, J. S. Speed, M.D.; Associated Editor, Robert A. Knight, M.D.; Third Edition, 1956. Volumes I and II. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10" x 7", pp. 3297, with many illustrations. Price: £22.

THIS third edition of "Campbell's Operative Orthopaedics" is a most comprehensive surgical text-book. The original edition set out to supply a comprehensive reference book of surgical techniques for the interne, the industrial surgeon, the general surgeon, and especially for the orthopaedic surgeon.

The preface to the first edition clearly states that orthopaedic surgery is not a mechanical equation and that the author evaluates the list of methods by his own experience. This evaluation is emphasized a little more in this last edition, but the book could be dangerous in the hands of a surgical tiro without the benefit of the wisdom of experience or considered advice from someone more senior.

For the experienced orthopaedic surgeon this book is an essential tool. This edition begins with a completely new and excellent chapter on surgical physiology. The remainder of the book follows the earlier pattern, but with the addition of the most important developments, and there have been many, that have occurred in the last seven years. After the war orthopaedic surgeons were stimulated to reconsider many old problems. Backache appeared to be on the way out, also bone infection. A few enthusiasts were being more radical in their treatment of tuberculosis of bone. Küntscher intermedullary fixation was seen first by the allies. In 1949 many of these things were treated in the second edition. This was very different from the pre-war first edition in 1939. Now many of these methods have been accepted, the orthopaedic surgeon's armamentarium being broadened. The author accepts Küntscher's clover leaf nail for certain types

of fractures. He also accepts radical excision of tuberculous granulation tissue as indicated sometimes. Thus many alternative methods are listed; most are American, and are techniques which the post-graduate, working for his board examinations, might see in the American orthopaedic clinics.

The editors regret that the "foreign" orthopaedic literature has not been completely recognized, but such important principles as Charnley's arthrodesis by compression have been considered at length with reference to the knee joint. It seems a pity that his central dislocation operation for the hip has not been included. The antero-lateral decompression operation used by Capener for the management of the tuberculous paraplegic is another important British contribution that has been added to this edition.

In loyalty to the great American, Smith-Petersen, the section on the cup arthroplasty of the hip is now written by Otto Aufrank, who is still performing the cup operation in Boston. He is not satisfied with the original method and has made several major alterations in the technique. This is well written and included in the chapter on arthroplasty of joints. The other methods of hip arthroplasty with use of the acrylic and metal prostheses are included in the chapter on delayed union and non-union of fractures of the neck of the femur.

This comprehensive orthopaedic reference book is valuable for the critical surgeon specialist.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Course in Practical Therapeutics", by Martin Emil Rehfuess, M.D., F.A.C.P., LL.D. (Hon.), and Allison Howe Price, A.B., M.D.; Third Edition, 1956. London: Baillière, Tindall and Cox, Limited. 11" x 9", pp. 989, with illustrations. Price: 120s.

The first edition (1948) and the second edition (1951) have been reviewed in this journal. There are 24 contributors to this edition.

"The Medical Annual: A Year Book of Treatment and Practitioners' Index", edited by Sir Henry Tidy, K.B.E., M.A., M.D. (Oxon.), F.R.C.P., and R. Milnes Walker, M.S. (Lond.), F.R.C.S.; 74th year; 1956. Bristol: John Wright and Sons, Limited. 8½" x 5½", pp. 616, with 37 plates.

The general plan of this well-known and recurring production is unchanged.

"Proprioceptive Neuromuscular Facilitation: Patterns and Techniques", by Margaret Knott, B.S., and Dorothy E. Voss, B.Ed.; Foreword by Sedgwick Mead, M.D.; 1956. New York: Hoeber-Harper Book. 10½" x 7½", pp. 146, with illustrations. Price: \$5.50.

The aim of this book is to make available the basic information necessary to the learning of the "techniques of proprioceptive neuromuscular facilitation".

"The Support of Medical Research." A symposium organized by The Council for International Organizations of Medical Sciences. Established under the joint auspices of U.N.E.S.C.O. and W.H.O. Edited by Sir Harold Himsworth and J. F. Delafresnaye, C.I.O.M.S.; 1956. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 182. Price: 17s. 6d.

This book is the report of a conference held at the Royal Society of Medicine, London, from October 4 to 8, 1954.

"Studies in Toxectomy", edited by Nolan D. C. Lewis, M.D., Carney Landis, Ph.D., D.Sc., and H. E. King, Ph.D.; 1956. New York and London: Grune and Stratton, Incorporated. 9" x 6", pp. 256, with illustrations. Price: \$6.75.

There are 31 contributors to this book, which deals with psychosurgery as a form of therapy.

"Management of Life-Threatening Poliomyelitis: Copenhagen, 1953-1956, with a Survey of Autopsy Findings in 116 Cases", edited by H. C. A. Lassen, M.D.; 1956. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. 190, with illustrations. Price: 22s. 6d.

There are 22 contributors to this book.

The Medical Journal of Australia

SATURDAY, NOVEMBER 17, 1956.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE OLYMPIC GAMES.

THE visit to our shores of a concourse of high-ranking athletes, prepared to contest for world titles, must arouse deep interest over a wide field of responsiveness. The members of the medical profession, ever sympathetic towards clean, healthy recreation, will not be lacking in appreciation of the purely sporting aspect of this great event, but they will also manifest a vocational curiosity in the anatomical and physiological factors which lead to success. It has been brought as a reproach against medical men that they are more interested in disease than in good health, but actually no one is more appreciative of the smooth working of the body's mechanisms than the man who has given time and thought to their study. This band of youthful athletes with their superb physique and grace of action will arouse both professional and artistic admiration. The Greek origin of the function will be remembered, and in the profession there will be the same veneration for the Greek striving after perfection in the human body; it will not be forgotten that the site of the games has the same latitude south as Athens has north.

The physiology of athletic effort is extraordinarily complex, and here it may be confessed that the methods used in training for a competition are largely empirical; certain exercises have been found to be useful irrespective of approved scientific theory which comes later, giving precision to what had been discovered by trial and error. Each department of the body's living machinery called into operation in a sporting contest presents us with problems of essential character, many of which have not been fully subjected to scientific investigation. The mechanics of a

muscle's origin and insertion, the disposition of its contractile fibres and their attachments to sinew and tendinous intersections have all been examined in detail; the chemical and thermodynamic happenings in a contraction have occupied the attention of a large number of investigators, and the story has not yet been fully told. There are, however, many other considerations which await examination; for example, do muscles in a reciprocating movement, and most unskilled limb movements are reciprocating, display anything resembling what was early found to be necessary in the steam engine? Does a muscle go into activity before the movement produced by its antagonist is completed, and does a muscle cease to be active before its "stroke" has finished? Again the circulation of blood through muscular tissue is beset with curious problems; the same capillary which brings the correct amount of oxygen must also remove the correct amount of carbon dioxide—obviously the oxidizable materials proffered to the contractile fibre must be in fixed ratios. A particularly important consideration, and one often forgotten, is that between capillary blood vessel and muscle fibre there is a layer of tissue fluid or lymph through which all interchange of oxygen, fuel, carbon dioxide and other exhaust substances must take place. It is obvious that the thinner this layer is the quicker will be the necessary transfer. This explains one of the benefits of training and is the main advantage arising from massage. A joint has a passive role to perform, but it, too, manifests features demanding consideration, such as the limits of movement imposed by ligaments and the critical thickness of the articular cartilage which will allow plasma nutrition by diffusion from adjacent bone blood vessels.

It is generally conceded by those physiologists who have studied athletic performances that it is the circulation which plays a special part. Myocardial mass and efficiency of contraction, capacity of the ventricles and the completeness of systolic evacuation have all been considered, but there are other problems. One is apt to forget that the coronary arterial circulation is diastolic, and hence nor-adrenaline, which raises the diastolic pressure considerably and the systolic pressure but little, must come into operation. Again if one ventricle was to eject consistently a very little more or a very little less than the opposite ventricle, then in a short time all the blood would be heaped up in either the pulmonary or systemic system. To expect absolute equality of output is unreasonable; what apparently happens is that if there is an accumulation on one side, the law of Starling comes into action and the excess is ejected—possibly the pericardium plays some part here. Professor A. V. Hill, once a Cambridge athlete, has made it clear that the most energetic action of lungs and heart cannot supply the oxygen required by the body in violent exercise. Fortunately the muscle can still continue to contract powerfully despite an oxygen debt of which lactic acid is the index. When the exercise is over the debt is redeemed by dyspnoea.

When all the physiological mechanisms of the body have been taken into consideration there remain the imponderables, and the imponderables will often determine victory or defeat. The stimulus of competition and of the presence of a large and responsive audience must be regarded as factors of consequence in deciding the issue

of each contest, and here we are in territory which is partly physiological and partly psychological. Fear may weaken a competitor's strength and tenacity. More than once has a pugilist arrived at the ring a beaten man.

The Olympic Games are worthy of deep consideration by Australians from another standpoint than sport. A large number of visitors from all over the world will make the games a good pretext for a survey of our Commonwealth. There are certain shortcomings which we cannot rectify in the time; our hotels, sleeping cars and breakfast grape fruit may not be up to American standards, and, of course, there is the deplorable inconsistency in our railway gauges, but our air transport needs no apology. One thing we can do and ought to do is to show every courtesy in our power to our visitors, who, if so treated, will return to their home lands as unofficial but influential and successful ambassadors of goodwill. The medical profession needs no exhortation in this matter; it can be confidently expected to be in the lead, no matter how the athletic victories are shared, in making the visitors feel they are welcome and are liked. Hopes will be expressed that they may come again even if there is no other inducement than to see this continent and its many unique attractions and its friendly people.

Current Comment.

TWO MYSTERIOUS DEATHS IN 1712: THE DUKE AND DUCHESS OF BURGUNDY.

THE last years of Louis XIV were far from happy. France had suffered badly from the wars in which she had been involved; Colbert's plans for the financial, commercial, industrial and naval reorganization of the country, evolved in the early days of the reign, had been ruined; opposition to the régime of *le roi-soleil* arose; and a series of deaths in the royal family brought the king great personal sorrow. The Dauphin, his eldest son, died in April, 1711; his eldest grandson, the Duke of Burgundy, and also the Duchess of Burgundy, died in strange circumstances in February, 1712; and their eldest son, the Duke of Brittany, died in March, 1712, at the age of five years. The king himself died in September, 1715. Both the Duke and the Duchess of Burgundy died after acute illnesses lasting for several days. There has never been any certainty as to what the disease was. One firmly held opinion was that they were poisoned; the most favoured diagnosis is measles. André Jobard¹ has gone into the question again, and advances a completely new proposition. The story that he tells and the clinical details that he presents to support his diagnosis are of considerable interest.

The Duchess, "the darling of the Court,"² was the first to fall ill. On January 18 her face became inflamed; but this condition cleared up in two days. The Duchess, like everybody else at that time, had very bad teeth. On the evening of February 5 she took to her bed with violent shivering and fever. She rose next day, but had a similar attack in the evening. On February 7 pyrexia was still present, and at six o'clock in the evening she began to suffer from temporal headache, very violent and localized to an area the size of "*une pièce de six sous*", as Saint-Simon put it. No treatment gave the unfortunate patient any relief from her headache; she was given tobacco to smoke and to chew, and opium, and was bled twice from the arm, all to no avail. The headache lasted until February 8, and during that night mental confusion

appeared, with high fever, great prostration, and a few spots on her skin which gave rise to the hope that the disease might be measles, an epidemic of which was rampant at the time. However, this hope faded with the disappearance of the rash during the night of February 9. An emetic was given, with no particular success. On February 11 it was decided to administer the sacraments. That night there was a consultation of seven doctors; Saint-Simon records that they all agreed that the Duchess should be bled from the foot before the fever had a chance to increase, and that if that measure was not successful, an emetic should be given towards the close of the night. On February 12, in spite of this vigorous treatment, the Duchess's weakness increased, and she was conscious only for short periods. She died in the night, after seven days of illness.

The Duke of Burgundy, now the Dauphin, had remained with his wife from the onset of her illness until the day before her death; on that day, he himself was overcome by the disease and had to take to his bed. Saint-Simon saw him on February 13 at Marly, where he had gone in order not to be present at the preparations being made at Versailles for the Duchess's funeral—the custom of the time. Saint-Simon was struck by the change in the Duke's appearance—his eyes were unduly bright and there were livid patches on his cheeks. However, he was walking up and down his room, and even went off to see the king, who also was at Marly. Louis, alarmed at the appearance of his grandson, ordered that his pulse should be felt by the doctors who were present, and sent him off to bed. On the next day the Duke's condition was worse, and he confided to Boudin, who had formerly been his wife's chief physician, that he believed he had been poisoned. (Boudin some time previously had warned him about a possible attempt to poison him.) On February 15 and 16 the fever rose higher and higher, and the pulse was faint, thready and difficult to feel; the livid patches spread all over the patient's body. The anxious watchers tried desperately to believe that the disease was measles, but without much hope, because of the Duchess's illness and death. On the next day the patient's condition seemed hopeless, and he died on February 18 at eight o'clock in the morning, after an illness lasting seven days—the same as that of the Duchess.

Autopsies were carried out on both bodies. In that of the Duchess, the only lesions found were in the brain, at the level corresponding with the site of her violent headache. Fagon and Boudin decided that she had been poisoned; Maréchal would not agree, insisting that he had performed many autopsies in which he had found similar lesions, without there being the slightest possibility of poison in any case. (Jobard comments that Maréchal, in the absence of any positive findings, preferred out of the kindness of his heart to brush aside any suggestion of poisoning, in order not to cause the king unnecessary pain. His colleagues were not so circumspect.) The autopsy on the Duke showed that "*les parties nobles*" were reduced to pulp; "his heart no longer had any consistency; its substance dripped to the ground between their hands". (Jobard remarks that these are the characteristics of myocarditis.) However, no abnormality was found in the brain. The same opinions were given—Fagon and Boudin for poisoning, Maréchal against it. Maréchal's diagnosis is interesting:

The poison which caused their deaths . . . was a natural venom arising from the tainting of the whole blood-stream, inflamed by a high fever which seemed less because it was internal; from that came the corruption which ruined the organs.

At the present time, as Jobard remarks, this would be given the name of septicæmia; and after much thought and study, he has come to the conclusion that the royal victims perished from an epidemic infective fever.

Turning first to the clinical history of the Duchess, Jobard eliminates the idea of septicæmia resulting from a dental infection. As he points out logically enough, although this diagnosis would suit her case, it would make it necessary to accept the position that two different fatal

¹ *Presse méd.*, June 23, 1956.

² *M. J. AUSTRALIA*, March 3, 1956.

infections carried off husband and wife almost simultaneously. That is possible; but the solution on which he has decided fits all the circumstances much better. The question then arises, what was the fever? It was obviously one not known at that time, at least by the majority of doctors; it was not smallpox or chicken-pox, whose clinical manifestations were familiar; it was almost certainly not measles, for the catarrhal prodromata were missing; nor was it scarlatina. It does not fit with the characteristics of typhoid, which has an insidious onset. There remains typhus; the vermin which crawled about under the tall periwigs worn at the time must have helped it to spread. The characteristics of the illnesses under discussion that are in favour of a diagnosis of typhus are as follows: the sudden onset, the rapid course, the mental confusion, the headaches, and the mysterious rash which made such an impression on the medical attendants. Although typhus was apparently first described by Jerome Cardan in 1536 under the name "*morbis puticaris*", the clinical description of the disease as an entity given by Frascatoro in 1546 is much better known and much superior. Frascatoro based his description on his observations of an epidemic in Italy. Typhus was described as a new disease in England in 1643 by Sir Edward Graves. It is not certain that it existed before the twelfth century. Jobard believes that in France in the seventeenth century the disease was not recognized, and was confused with the other eruptive fevers. To meet the possible objection that typhus is rare in winter, he points out that there have been some famous epidemics in that season; an example is that which decimated the French army in 1813, after the retreat from Germany. He believes that at the beginning of the eighteenth century, in view of the prevailing conditions of hygiene, the disease must have smouldered constantly in endemic-epidemic form. Moreover, an endemo-epidemic of typhus could very well develop alongside an epidemic of measles, which was a common occurrence at that time of the year. The fact that typhus was not a clinical entity might cause confusion between the two conditions, which may have one or two signs in common. The light cases which ended in recovery did not shake the doctors' confidence in their mistaken diagnosis, and the rapidly fatal cases (unusual in an outbreak of measles, which is dangerous only when pneumonic complications supervene after a long period of illness), were branded "malignant measles". That satisfied everybody, for, as Jobard cynically remarks, is not the important thing to die according to the rules? The conclusion, with all necessary reservations, is that the Duke and Duchess died from typhus. However, if purely medical considerations are left on one side, the idea of poisoning has some points in its favour. The Duke of Burgundy, who had been the Dauphin since his father's death, stood in the way of one or two people. The Duke of Orleans had his eyes on the regency, and the Duke of Maine, who had been legitimated, may have hoped to succeed Louis XIV. There is an odd story concerning a Spanish tobacco box presented to the Duchess on the day when she fell ill; it was given to her by the Duke of Noailles, the tool of Madame de Maintenon, whose favourite was the Duke of Maine. The box mysteriously disappeared after the Duchess had taken some of the tobacco which it contained.

Jobard ends his interesting account of this tragic page of history by advocating adherence to known facts and the avoidance of undue speculation. This attitude leads him to the conclusion that the Duke and Duchess of Burgundy were done to death by a rickettsia.

THE NORMAL BLOOD PRESSURE.

No agreement has ever been reached on the standard values beyond which the systolic or diastolic blood pressures could be considered to be abnormal, and to be indicative of cardio-vascular disease. In recent years the importance of stress in causing both temporary and permanent elevation of the blood pressure has been increasingly appreciated. In young persons the blood

pressure may remain at high levels for considerable periods and then return to normal average values when the stress is removed. It has been suggested that young people with a labile blood pressure may well, in later life, develop a permanent hypertension. It has been generally accepted that the diastolic blood pressure is the more useful guide to vascular disease, though even here it has so far proved impossible to lay down precise criteria of normality. Sex and race appear to have little or no influence upon the blood pressure, and the influence of age in causing normal variations is not so plain as had previously been thought.

In an attempt to obtain a satisfactory assessment of the normal ranges of blood pressure, R. El. Boynton and R. L. Todd¹ examined the formidable number of 75,258 university students. Between the second and the fifth decades of life there was surprisingly little difference in the men. The average systolic blood pressure in the males was 122 millimetres of mercury; the average for women was lower, but tended to rise with age. The mean diastolic blood pressure in the males was 74.5 millimetres of mercury; again the value tended to be lower in women but to rise more with increasing age. From this survey it was not possible to lay down criteria of normality, but below the age of forty years comparatively few patients at any age had a systolic blood pressure above 140 millimetres of mercury, and the majority of men were to be found in the range of 110 to 129 millimetres.

A. M. Fishberg² suggests that the divergences of opinion on the normal values result from differences in the concept of normality. It seems likely that the upper limits of normality should be set at 140 millimetres of mercury, systolic, and 90 millimetres, diastolic. The higher the blood pressure above these levels, the more likely is the patient to develop hypertensive or arteriosclerotic disease. Especially in the older person, the average group blood pressure levels will be raised by those individuals who have mild hypertension. The most common blood pressure is not necessarily the ideal one. Concepts of normality are dependent on the degree of diminished arterial elasticity which can, with advancing age, be regarded as normal. Fishberg considers that elevation of blood pressure by apprehension is not insignificant. The patient with an emotionally labile blood pressure is more prone to develop subsequent hypertensive disease. Young patients with incipient hypertension often have ophthalmoscopic signs and are usually large, strong boys with at least one hypertensive parent.

From their own criteria of normality, R. S. Palmer and H. M. Muench³ suggest that from 10 to 15 million Americans are suffering from some form and degree of hypertension. They made long follow-up studies of a large number of patients with essential hypertension and found that the prognosis could be related to the initial degree of hypertension. Risk was particularly high in the younger patients. It was generally greater in men, and the hypertension tended to increase with advancing age. Except in the severe degrees of hypertension the duration of the abnormality did not appear to affect the prognosis.

The most recent study of variations in blood pressure in young, healthy adults is that of N. Szent-Györgyi.⁴ The survey was carried out on 3508 students at the University of Chicago. All students with organic disease which might influence the blood pressure were excluded from the survey. The normal high values were taken as 140 millimetres of mercury, systolic, and 90 millimetres of mercury, diastolic. Students with levels above these were examined again later, and their social and racial backgrounds were investigated. On these criteria, 7.7% of the males and 3.2% of the females were found to be hypertensive. American men had an incidence of hypertension considerably greater than that of non-American men, despite the fact that the foreign-born men tended to be under more stress and tension at the time when the survey was con-

¹Arch. Int. Med., October, 1947.

²M. Clin. North America, May, 1954.

³J.A.M.A., September 5, 1953.

⁴Circulation, July, 1956.

ducted. However, this geographical factor was related entirely to the low incidence of hypertension in white students whose homes were in neither Europe nor America. The incidence of hypertension was far higher in American Negro students than in American white students. Again hypertension was as common in Americans of Oriental, as in Americans of European, origin, but in Asian students the incidence of hypertension was much lower. Of all the hypertensive students the majority of the men were below the age of thirty years, but the majority of hypertensive females came from the relatively small group of women above this age. An interesting point raised was that, after some years' residence in America, foreign-born students appeared to have the same chance as native Americans of being hypertensive. The relatively small proportion of foreign students means that caution must be used in the interpretation of these findings. However, it does appear that in North America and in Europe there are environmental factors which predispose to the development of hypertension both in the white population and in those of Asian or Negro origin.

THE EMERGING SCALP.

BALDNESS is one of the more cruel cosmetic changes associated with advancing age. In women the condition is relatively rare and is easily concealed; for men fashion denies its satisfactory concealment, and abandons them to the conscious shame of the emerging scalp. There can be fewer conditions in which popular supposition is so much at variance with the facts. Far from being a true sign of senility, baldness particularly affects the strong and the vigorous. Some of the best athletes appear to lose their hair at an early age, and the bald at least assert that the lazy and the mentally dull usually have a luxuriant thatch. Nevertheless, baldness causes a great deal of unhappiness, absorbs much time and thought in its concealment, and lines the pockets of unorthodox practitioners and drug firms with something more substantial than hair. The factors causing baldness are unknown. It is apparent that male hormones and heredity have some influence, but the rest is mostly conjecture. E. J. Moynahan¹ states that scalp hair grows faster than any other hair, and the rate of growth is higher in women than in men and children. Hairs, like nails, grow faster in the warmer months of the year. Cutting or shaving the hair—and this applies generally—neither encourages hair to grow nor increases its growth rate. There is no known method which will induce hair to grow on bald areas devoid of hair follicles. Slight recession of the frontal hair margin is normal in girls at puberty, though the amount of fronto-parietal recession which may be regarded as physiologically normal for the male is unknown. It is apparent that the pattern of hair loss on the scalp is a "human" characteristic, as it occurs not only in senile male baldness, but also as a normal finding in neonatal life, and in the conditions such as myxoedema or masculinizing tumours in the female, and it was observed in survivors of all ages after the atom bomb explosions in Japan. Moynahan does not support the recent revivals of the theories that baldness is produced either by ischaemia of the scalp or by the seborrhoeic diathesis. Congenital alopecia usually arises as a genetic mutation, though it has been suggested that intrauterine events may produce hypotrichia and other ectodermal defects. Monilethrix, in which the beaded hair breaks off at one of the constrictions and rarely exceeds one-quarter inch in length, is also an inherited condition. The treatment of this and allied conditions of the hair has been unsatisfactory, though the author reports some early success with the use of ACTH. Alopecia may also be associated with other congenital ectodermal defects, such as absence of the sweat glands and abnormalities of the nails.

When baldness runs in families it may affect both sexes, although the hair loss in women is less pronounced.

¹ M. Press, July 25, 1956.

The baldness is not attributable to a sex-linked gene, but is provoked by the male sex hormones. There are probably several genetically different types of baldness.

The cause of *alopecia areata* and its more severe forms is also unknown. In most cases the patients recover spontaneously after some months, and no treatment has any effect except for the corticoids which, if given in sufficient doses, will produce rapid regrowth of the hair on the bald patches; this hair may fall out if the corticoid is prematurely withdrawn. Hydrocortisone ointment is quite useless in this respect.

A variable degree of hair loss may follow severe infections and haemorrhage, though regrowth is usually complete. The same phenomenon is sometimes seen in the normal post-partum period. Thinning and loss of dull, brittle scalp hair may be seen in both myxoedema and hypoparathyroidism. Virilism in women is always accompanied by some degree of baldness.

Massage of the scalp, particularly if it is at all vigorous, will sometimes lead to partial baldness, as the hair in individuals attempting this form of therapy is usually already fine and brittle. The same effect may be produced by vigorous brushing. In women the traction of curling pins may also result in frontal and temporal hair loss.

Moynahan concludes by making some comment on the care of the hair. It should be brushed and combed night and morning until all tangles are straightened out. The bristles of the brush and the teeth of the comb should not be sharp, and both should be washed frequently in soap and water. Frequent cutting can have no good effect, and the only effect of singeing is detrimental, as the keratin in the hair shaft is burned, resulting in brittleness and fragmentation. The wearing of hats has no influence on the hair; shampooing and washing should vary with the individual need. Hair waving is harmless if performed skilfully, and if the hair is not too fine. "Cold perm" lotions should be used carefully. Excessive bleaching leads sooner or later to partial alopecia, and hair dyes may occasionally produce dermatitis and temporary alopecia. Moynahan suggests that the aetiology of several types of baldness may well best be studied by the general practitioner.

THE AETIOLOGY OF MONGOLISM.

MONGOLISM is one of the least understood of the primary amentias. Various views have been expressed on the basal defect in this condition, and it seems not unlikely that the cause of the clinical features is bound up with an intrauterine panhypopituitarism. However, the cause of this glandular failure, if such it is, remains completely obscure. The interposition of a healthy mongoloid child into an otherwise normal family is an event which calls for a good deal of patient courage and forbearance. The real importance of mongolism is to prevent it if this is possible and to remove, if this is justifiable, the stigma of hereditary abnormality which affects the relatives of the mongoloid child. It is generally accepted that mongols tend to be either at the younger end of a large family, or the children of elderly parents. No clear-cut hereditary factor has been demonstrated, though some authors suggest there is some increased likelihood of further mongol children in an afflicted family. With the object of throwing light on the clinical picture and the aetiology of mongolism, J. Øster² has made a statistical survey of a large unselected section of the Danish population containing 526 living mongols, all of whom were examined; a family and genealogical history was compiled for each. In addition, statistics of mongol births were used in the calculation of incidence. It was found that the sex ratio was normal, and the mongol births were distributed throughout all social classes with the greatest frequency in the independent pro-

² Danish M. Bull., August, 1956.

professional classes, in whom marriage and conception take place at a relatively late age. The average age of mothers of normal babies was 28.7 years and of mongol babies was 35.1 years. There was one mongol for each 618 live births. The incidence of mongol births increased progressively with the age of the mother from 0.061% in the youngest group to 1.78% in the oldest group of mothers. When the age of the mothers with one mongol child was taken into account, there was no subsequent increase in the incidence of further mongol children in the same family, neither was there a familial accumulation of the defect in more distant relatives. There was no increase in the numbers of first cousin marriages in the parents of mongols. There was no increased incidence of any of the mental diseases, with the exception of nervousness and neurosis, which appeared to be increased, especially in the mothers. There was also some increase in the incidence of deaf mutism in the siblings of the mothers and of thyrotoxicosis in the female relatives, though these are not statistically significant. Study of the mothers leads to the conclusion that their reproductive faculty was reduced, and the onset of the menopause was rather late. Gynaecological disease seems to have been particularly frequent in the period before and after the mongol pregnancy, so that 15% of the mothers underwent curettage about this time. However, the menstrual cycles and the subsequent mongol pregnancy appear to have been normal. It is thus apparent from these studies of Øster that mongolism is in no way hereditary, though various exogenous maternal factors play a significant part in the occurrence of mongolism. Of these factors, the most important appear to be the advanced age of the mothers and their depressed reproductive faculty. Øster suggests that increased family planning, by reducing the numbers of children born to older mothers, should bring about some reduction of the incidence of mongolism. It would appear that 30% of the mongol births would have been avoided if children had not been conceived by women over the age of forty years.

THE BRITISH EMPIRE CANCER CAMPAIGN.

THE voluminous report of the British Empire Cancer Campaign covering the activities during 1955 of a vast number of investigators in universities, research institutes, hospitals and the like throughout the Empire has just been published. It is the thirty-third annual report. Clearly if £400,000 has been spent on cancer research, the volume of investigational results must be large and a wide diversity of methods of approach and of techniques employed will be displayed. It is possible that some readers will be disappointed to find that the central mystery of cancer remains elusive, but he would be a presumptuous critic who denied the possibility that any one of the findings described may be of service in the ultimate triumph of the quest. A more serious criticism is that some of the promising discoveries set out in previous years have not led to supplementary investigations. Perhaps the silence of this report on such matters may be due to the highly commendable attitude of withholding publication until fully proved conclusions have been reached. This is the case with Professor Green's hypothesis of an immunological basis for cancer which appeared last year and was singled out for special mention in the review published in this journal.

The relation of cancer to smoking has been further investigated. Statistical methods favour the idea of causation, but there is evidence of something else in cities, an "urban factor", beside tobacco, which can be in operation. Experimental research on this problem has not yielded positive results. Most interesting is the exposition of differences in various countries. In males in Uganda there is a great preponderance of cancer of the penis, liver and skin, but a low incidence of malignant disease in the respiratory tract; in females cancer of the uterine cervix is common, but of cancer of the breast and respiratory and alimentary tracts there is little. In Eire

some strange manifestations are disclosed; in rural districts inadequate efforts to detect and to counteract tuberculosis in cattle have led to much phthisis in extrapulmonary tracts. The remarkable conclusion is reached that there is a close inverse correlation between cancer and tuberculosis. This was proclaimed years ago by the late Professor T. Cherry, of Melbourne. The report here mentions "the earlier concepts of Cherry", but there are no bibliographical references; the index gives the name of C. P. Cherry, of Cambridge, seven times, but no other Cherry appears.

Even if the future showed no advance referable to this report and this, as stated, dare not be postulated now, one should concede that a large number of young researchers are being trained in the very latest methods of applied physics, chemistry and microscopy, and this is all to the good. Some admirable new techniques are described, particularly the quantitative assay of minute amounts of aminoacids and of oxygen, whilst an ingenious method has been devised for demonstrating the histochemical localization of enzymes which will certainly be put to good use in other fields of inquiry than cancer research. The separate reports are preceded by an admirable summary of the main items, but the reader is advised not to be content with this abstract, but to study the detailed records, at least in those subjects which interest him.

COLOUR VISION IN NATIVE RACES OF AUSTRALASIA.

UNTIL recent years little or no work has been done on the comparative anatomical and physiological characteristics of the various intermingled native races inhabiting the northern part of the Australian continent, and the numerous tropical islands which lie close to the mainland mass. The difficulties involved in the conduct of surveys are considerable. Geographical barriers and the frontiers of custom, understanding, and language are formidable. However, recent surveys have been made of such characteristics as blood groups, and now I. Mann and C. Turner¹ have made a survey of the colour vision in native races in Australasia. They comment on the old belief that colour vision is less highly developed in primitive peoples, and that many pigmented people have been considered to be insensitive to blue. It appears that native languages are poorly provided with words specific for different colours; hence the old belief that the colours were not perceived. Brightness and saturation are of more importance to native races than the actual colour. However, Mann and Turner report that workers amongst Pacific native races have found little evidence of defective colour vision except in such immigrant populations as the Indians in Fiji. The tests carried out by Mann and Turner involved interpretation of the Ishihara test cards. The authors first report on their findings in a sample of the white population. The numbers with defective colour vision in males varied from 12% in the Kimberly area to 4.15% in the Eastern Goldfields. It is suggested that the difference is largely due to occupational pressures upon colour-blind males. The white overall female incidence of 0.61% is also high, but the population sample was small in this group. The half-caste coloured males of the Kimberleys, Eastern Goldfields and Papua were, on an average, 3.2% colour defective; no defects of colour vision were found in the half-caste coloured females. Aboriginal males of the Kimberleys, Eastern Goldfields, and Papua were 1.9% colour defective, and only one aboriginal female out of 3201 was colour blind. Native races thus appear to have better colour vision than the white population of Australasia. However, marked exception to this was found in the isolated Marshall Bennett Islands, where 4.1% of the males were colour blind, and in the Trobriand Islands, where 6.5% of the males were colour blind. It is suggested that these isolated pockets of defective colour vision may be due to inbreeding.

¹ *Am. J. Ophthalm.*, May, 1956.

Abstracts from Medical Literature.

DERMATOLOGY.

Nickel Dermatitis.

C. D. CALMAN (*Brit. J. Dermat.*, July-August, 1956) states that nickel is by far the commonest sensitizing agent encountered in routine dermatological practice in London. In a study of 400 women patients, it was found that over 95% of them had stocking suspender dermatitis as their first manifestation of nickel sensitivity. In 19 cases the dermatitis initially was on sites in contact with the following: ear-rings, wrist-watch, brassière clips, necklace clips, spectacle frames. The general pattern of this dermatitis may be divided into (a) the primary eruption or direct metal-contact sites and (b) the secondary eruption or areas of spread, apart from metal contact sites. The primary sites are very numerous; some of these are situated in relation to suspenders, brassière clips, ear-rings, necklace, corsets, scissors, knitting needles, pins, hair grips, zip fasteners, brooches, spectacle frames, watches, bracelets, shoe buckles and perambulator handles. The secondary sites are as follows: elbow flexures, eyelids, sides of neck and face, thighs (inner aspect) and generalized. The secondary eruption behaves like a hematogenous spread similar to the ide phenomena in ring-worm and other infections. It is usually symmetrical and is related to the activity of the primary site. Nickel sensitivity induced by metal contacts at sites other than in contact with suspenders does not appear to cause a secondary eruption so easily. Nickel sensitivity can be confirmed by patch testing with 1% or 2.5% nickel sulphate solution; 5% or 10% solutions are unnecessarily strong and may give false positive results. Allergy to nickel is almost certainly permanent. Multiple contact sensitivity is not very uncommon. Patients with only primary sites affected settle down easily when metal contacts are removed. Once secondary spread has occurred, the prognosis is not so good. A high proportion of the patients had other varieties of eczema not specifically associated with their nickel-sensitivity pattern, especially on the hands.

Treatment of Herpes and Chicken-Pox with Immune Globulin.

J. C. RODANTE AND B. H. WILLIAMS (*Arch. Dermat.*, June, 1956) state that immune globulin was administered to 11 patients with severe herpes zoster. Prompt persistent relief from pain was obtained, and the skin lesions were modified. The authors suggest that immune globulin may minimize posterior root ganglion and spinal cord damage and thus prevent post-herpetic neuralgia. Since it is probable that herpes zoster and chicken-pox are all caused by the same virus, immune globulin was administered to five children with chicken-pox during the first day of the eruption. Examination of the skin lesions suggested that chicken-pox could be attenuated

in this manner. Large doses of immune globulin should be given over a period of four days to patients with severe herpes zoster and over a three-day period to patients with chicken-pox.

Cortisone and Lichen Planus.

P. B. MUMFORD AND J. K. MORGAN (*Brit. J. Dermat.*, July-August, 1956) report that ten patients were given cortisone by mouth, and ten acting as controls were given dummy tablets indistinguishable from actual ones. A standard dosage was employed—namely, 150 milligrammes (six tablets) daily for two weeks, followed by 75 milligrammes daily for a further two weeks. All patients were questioned on the first visit about personal and family histories of chest disease, peptic ulceration, diabetes and psychoneurosis. After the trial assessment was as follows: (a) with active tablets the response was good in six cases, fair in four, and poor in none; with dummy tablets it was good in one, fair in two, and poor in nine. The authors state that cortisone merely suppresses symptoms and has no effect on the underlying mechanism of the disease. However, they contend that the great irritation and distress which sometimes occur are adequate grounds for the use of cortisone in selected cases, since the temporary results are good.

Diffuse Hair Loss Associated with Selenium Sulphide Shampoo.

R. W. GROVER (*J.A.M.A.*, April 21, 1956) states that the repeated use of a shampoo containing selenium sulphide for the treatment of seborrheic dermatitis was followed in six cases by loss of hair in varying degrees. In all cases the shedding of hair stopped when the use of the shampoo was discontinued, and in one of the cases the phenomenon was repeated several times. Hitherto the only common adverse symptom noted has been that of excessive oiliness of the scalp, which occurs in about 31% of patients using the shampoo. Contact dermatitis has also been reported. The author believes that the falling of hair exhibited by the six patients may represent a toxic effect of absorption of selenium ion and urges caution in the use of shampoos containing selenium.

The Mycoses as a Therapeutic Problem.

F. MINDZINSKI AND J. LIPSKE (*Brit. J. Dermat.*, June, 1956) state that owing to the prophylactic and therapeutic measures initiated in the post-war years in the campaign against the mycoses, the incidence of these infections is steadily decreasing. This is due to compulsory notification to the public health centres. The diagnosis of mycosis was always confirmed by clinical and microscopic examination and by means of Woods' light. There was a high incidence of *tinea capitis* amongst children from three to six years, which must be attributed to a greater susceptibility to infection at this age. Out of 374 cases of mycosis treated, there were 248 cases of *tinea trichophytica* of the hairy scalp, 72 cases of *tinea favosa*, 45 cases of *kerion celsi* and nine cases of isolated *onychomycosis*

trichophytica or *favosa*. Epilation by X rays was carried out on all patients with *tinea capitis*. After complete removal of all hairs the treatment consisted of painting the scalp with 5% tincture of iodine twice daily and applications of Wilkinson's ointment once daily; the head was washed every day with soap and water. No child was discharged until the results of three successive examinations of fragments of hairs and scales were negative for fungus. When no scaly particles appeared during six months, and the results of microscopic examinations were negative for fungus, the patient was regarded as cured.

UROLOGY.

A New Method of Relieving Upper Ureteric Obstruction.

N. S. R. MALUF (*J. Urol.*, February, 1956) reports two successful results with a new technique of dealing with upper ureteric obstruction by lower renal polar vessels. In these cases there was no intrinsic obstruction at all, and the lesion was a bulging, or herniation, of part of the renal pelvic wall between the lower polar vessel (or vessels) and the upper or normally placed vessels. After freeing (that is, mobilization) of the lower polar vessels and also of the pelvis and upper part of the ureter, the lower vessels are made to slide up the interior surface of the pelvis so as to close up the gap of the vascular bifurcation, and so prevent future bulging of the renal pelvis in this area. The adventitia of the arteries are sutured together with very fine chromicized sutures on an atraumatic needle. In this way the vessels are not sacrificed, neither is any break made in the continuity of the pyelo-ureteric junction. Nephropexy is an advisable adjunct, and pyelostomy for a time may be indicated, though not in patients with sterile urine.

Pyelectasis.

R. LICH, J. E. MAURER AND M. L. BARNES (*J. Urol.*, January, 1956) discuss ureteric obstruction at or near the uretero-pelvic junction. Many of these are considered to be due to the pressure of aberrant renal vessels or fibrous bands. However, in many cases, when these structures are removed, it is noticed that the distended pelvis does not empty easily down the ureter. In such cases, a good result may not follow, and years later a secondary nephrectomy may be imposed on the surgeon. In 15 cases the authors resected the entire uretero-pelvic junction region and studied the appearance of the canal, opened by longitudinal section. Nine of these specimens showed distinct valve formations, hindering distal flow of urine, six showed suggestive valve formation, and two showed marked thickening of the ureteric wall, due to increase of fibrous and muscular tissue, and leucocytic infiltration. In seven out of the 15 cases there was an aberrant vessel or fibrous band, but in no instance did extrinsic pressure cause the pyelectasis, though this certainly does occasionally occur. In addition to this series of 15 cases there were another six cases of

pyelectasis, in which operation was performed by the resident staff, making a total of 21 cases. The ages of the patients varied from six months to fifty-two years. Post-operative observation has extended from seven months to five years. There were only two failures, and these were considered to be due to poor judgement in selection for operation, rather than to any defect in the operation. In both cases, extensive pyelonephritis was present, and nephrectomy should have been chosen. In the technique of operation, the renal pelvis is partly cut away with scissors, along a curved line, so that the convexity faces the hilum. The ureter is cut across well below the uretero-pelvic junction, and its proximal end is split for two centimetres along its lateral aspect. The cut edges of the ureter are sutured to the lowest part of the renal pelvis, after the upper portion of the latter has been closed. No ureteric splint is used, and drainage is by nephrostomy for seven days and by an extrarenal tube for nine days.

Congenital Unilateral Multicystic Kidney.

H. M. SPENCE (*J. Urol.*, December, 1955) emphasizes the features of congenital multicystic kidney. He states that these warrant its classification as a separate entity to be distinguished from congenital polycystic disease, multilocular cysts and multiple simple cysts. True polycystic disease is almost invariably bilateral, and is ultimately fatal; the reverse is the case with multicystic kidney, in which there is an opposite normal organ, and the prognosis after nephrectomy is excellent. The term "unilateral multicystic kidney" was first proposed by Schwartz in 1936. The striking feature of this lesion is replacement of the entire renal parenchyma by many cysts, varying in size from that of a pea to that of a hen's egg, and held together in a form like a bunch of grapes by loose connective tissue. The ureter is characteristically absent or rudimentary. The vascular pedicle may also be rudimentary, or even absent. There is no evidence that the lesion is a variety of hydronephrotic (obstructive) phenomenon. In congenital polycystic disease, varying amounts of normal parenchyma, capable of function, are seen microscopically between the cysts. Multilocular cystic kidney shows the disease process in part of the organ only, and cysts are seen within cysts. The word "polycystic" should not be used loosely to denote either the multicystic kidney or the multilocular cystic kidney. Polycystic disease is congenital, bilateral and ultimately fatal. As regards etiology of multicystic kidney, all that can be said for certain is that it is a congenital defect resulting from foetal maldevelopment, not on an hereditary basis. In true polycystic disease there is, as well as congenitalism, an inherent genetic fault. The author reviews 15 cases previously reported in the literature and describes four cases of his own. On routine examination in a presumably healthy infant, a large, unilateral, non-tender, movable mass is found in one flank. Signs of pressure on the bowel and the symptom of pain are unusual. Cystoscopy and attempted retrograde study have

shown either absence of a ureteric orifice, or blind termination of a small ureter below the affected kidney. Excretion urography shows no function on the affected side. There is no predilection as to size or sex. Most cases occur in infants, but a few are in adults. In the differential diagnosis Wilms tumours and hydronephrosis are to be considered, but in such cases it does not matter if pre-operative diagnosis is not made, for the treatment common to all is surgical exploration and usually nephrectomy, the opposite kidney being found to be normal.

Surgical Experience in Orchidopexy.

R. E. GROSS AND T. C. JEWETT (*J.A.M.A.*, February 25, 1956) state that cryptorchidism is a common abnormality, and in recent years has given rise to much discussion regarding proper modes of therapy, optimum age for treatment, and results that can be expected. The authors have had a long and large experience, and in this paper they base their surgical experience on 1222 operations. They state that true cryptorchidism must be distinguished from high retracted testis, which is normal in some boys up to the age of puberty. In true cryptorchidism the testis cannot be palpated, or, even if it can be palpated, cannot be displaced into the scrotum. An undescended testis can produce enough androgen to fulfil its endocrine function, but is liable to mechanical injury and is unable to produce spermatozoa. The psychological need for correction is an important consideration; however, evidence that such correction reduces the danger of subsequent malignant change is unconvincing. Orchidopexy generally involves both repositioning of the testis and repair of an oblique inguinal hernia; in this series there has been no recurrence of the hernia. Operations of the Torek type, in which the testis is temporarily attached to the thigh, are likely to damage the testicular blood supply, and therefore to give poor results. The operation performed by the authors involves freeing the ductus deferens down to the base of the bladder, and the spermatic vessels well up to the lower pole of the kidney, so as to try to eliminate all tension. The operation is carried out on one side at a time, preferably at between nine and eleven years of age. Of a group of patients studied ten years or more after bilateral orchidopexy, 79% have been shown to be fertile.

Antimicrobial Agents in Urinary Infections.

R. D. HERROLD (*J. Urol.*, June, 1956) divides antimicrobial agents for urinary infections into two main classes: (i) those which act in an excretory capacity only; (ii) those which also act in this way, but in addition have a bactericidal effect in the tissues themselves. The author traces the history of, and indications for, the various sulphonamides and the earlier antibiotics. He states that "Chloromycetin" acts rather similarly to "Aureomycin" and "Terramycin", but that it is more effective against *Proteus*, especially when combined with streptomycin. He has found "Chloromycetin" singularly free from side reactions. Polymixin B is the only effective anti-

biotic for urinary infections that are derived from a bacillus rather than a fungus or a streptomyces. It contains a nephrotoxic factor; hence its use is limited. However, it is safely used when only one injection per day is given, concurrently with oral administration of "Terramycin" in some resistant infections, notably with *Pseudomonas*. Erythromycin, although derived from a streptomyces, is largely anti-coccal in action and is given when such infections are resistant to penicillin. Cycloserine ("Seromycin") is a new broad-spectrum antibiotic. While sometimes causing dizziness, it has low toxicity, no colonic side-reactions, no nephrotoxicity and no skin reactions. Since no resistance is developed to it, long-term therapy can be applied in some infections, notably pulmonary tuberculosis. Similarly, in chronic and recurrent infections of the urinary tract, cycloserine has proved of value if continued for long periods—for example, two months or so. This drug is widely applicable, the exceptions being for infections with *Pseudomonas*, *Proteus* and *Streptococcus faecalis*. It was noted that two patients with trichomonas infection of the prostrate and urethra were cured, after four and six weeks respectively, with cycloserine. It is also noted that the best combination of agents to fight *Pseudomonas* is that of "Terramycin" and streptomycin; and for *Proteus* a combination of "Chloromycetin" and streptomycin.

Prognosis of Primary Neoplasms of Renal Pelvis and Ureter.

J. E. DEES (*J. Urol.*, March, 1956) states, after reviewing recent literature on the prognosis of primary neoplasms of the renal pelvis and ureter, that it does not lend itself to statistical analysis. There are too many inherent variables in this complex subject. So it has been more profitable to select for discussion a few of the carefully studied, and followed, groups of patients. The results of treatment of 22 patients with primary tumours of the renal pelvis and the ureter are studied. There were ten transitional cell carcinomata of the pelvis, two squamous cell carcinomata of the pelvis (by metaplasia from chronic irritation), two benign papillomata of the pelvis and eight transitional cell carcinomata of the ureter. Four patients in the first group of ten are living at six, seven, eight and eighteen years after operation; five patients died within one year of operation, and the others within two years. Of the two patients in the second group, one died within two years; squamous cell carcinoma of the pelvis has a notoriously hopeless prognosis. The two patients in the third group are living at eight and fifteen years after operation; the prognosis of benign papilloma is relatively good provided complete nephro-ureterectomy is carried out. Of the eight patients in the fourth group, four were dead in less than two years after operation; four were known to be living less than one year after operation, but they could not be followed after that. The point about this group seems to be that carcinoma of the ureter which has invaded the muscle offers an outlook as bleak as that of squamous carcinoma of the pelvis.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on Tuesday, July 17, 1956, Dr. W. L. CALOV in the chair. The principal speaker was Dr. Peter Hall, honorary assistant physician to the hospital.

Clinical History.

The following clinical history was presented.

When first admitted to hospital in September, 1954, the patient, an electrical fitter, then aged thirty-three years, had complained of "recurrent colds" for six months. A chest X-ray examination showed an opacity in the lower lobe of the right lung. Thoracotomy was undertaken, and a carcinoma of the bronchus was removed by lobectomy. Microscopically the growth proved to be of the anaplastic squamous type. No growth was found in a hilar gland. Convalescence was uneventful, and he was discharged from hospital in mid-October, 1954.

He returned to work and gained one and a half stone in weight. However, starting in March, 1955, he suffered repeated attacks of pleurisy, mainly on the right side. These attacks were associated with cough, dyspnoea and fever. He had five or six attacks in all; some settled down with rest alone, whilst others were treated with sulphonamides and antibiotics. The later attacks were accompanied by a radiological "shadow" at the right lung base. In January, 1956, he developed a similar pain over the left lung base with cough and fever. This did not respond to antibiotics, and he was finally admitted to hospital on February 2, 1956. Apart from loss of one stone in weight he had no other symptoms.

Physical examination of the patient showed some consolidation at the left lung base with a pronounced friction rub. His temperature was 101° F. No other abnormalities were noted. His blood pressure was 135 millimetres of mercury, systolic, and 75 millimetres, diastolic, and a blood count at this stage showed a leucocytosis which persisted. A week later a bone marrow biopsy was reported on as follows: "Possibilities are a chronic myeloid leukemia or a leukemoid reaction; it is not possible to differentiate at this stage." An X-ray report at this stage was as follows: "No secondary deposits seen in the sternum, lumbar spine or pelvis. The texture of the bones in these regions appears normal."

During the next six weeks or so the pleuritic pain, which was often agonizing, appeared sporadically on both the left and right sides and there was an irregular but persistent fever up to 103° or 104° F. accompanied by drenching sweats and occasional rigors. On two occasions he produced blood-streaked sputum. Cortisone was given for two weeks (50 milligrammes twice daily) without effect, and many special investigations were performed. The results of these are listed chronologically in Table I.

On March 19 (one week before his death) a new component of his clinical picture appeared, for he now complained of pain and tenderness in the right hypochondrium, and this was accompanied by slight icterus. The liver became palpable, extending for three or four centimetres below the costal margin. Hepatic function was investigated (see Table I), and a liver biopsy was performed, the histological report on which was as follows: "Normal architecture; many neutrophils are present in the portal tracts and there are a few small foci of parenchymal degeneration. There is a non-specific hepatitis present."

During the last week, the patient's condition gradually deteriorated, he became semi-comatose, and he died on March 29, 1956, after eight weeks in hospital.

Clinical Discussion.

DR. P. F. HALL: I suppose that I should say at the outset that, from the information which is available, to my mind it is not possible to be dogmatic in making a diagnosis in this case. However, I expect from the nature of things that this is what one anticipates at such a conference. I have summarized the bare facts about this man's illness on the blackboard. We know that this man was an electrical fitter, but this does not tell us much. We do not know whether he resided or was ever engaged in active service outside this country or not. For a period of six months he had respiratory symptoms which apparently were not very severe, in September, 1954, he underwent a lobectomy, and in the

following six months he was relatively well. There was then a period during which his health deteriorated, and from that time to his death was about a year.

Let us take first the clinical picture and then the special investigations, which have been fully listed. We find that he was quite well until March of 1954, and thereafter suffered a series of "colds", which was apparently something unusual for him, and an X-ray film of the chest, which is not at the moment available, showed an opacity on the lower lobe of the right lung. Six months after symptoms began it was decided to explore his chest. At operation carcinoma of the bronchus was found, the lower lobe of the right lung was removed, and the growth histologically was found to be an anaplastic squamous carcinoma; at the time of operation no growth was found in the hilar lymph glands. He then underwent a convalescence which was not interrupted by anything remarkable, put on a good deal of weight, came back to what I assume was his normal weight, and returned to work, during the next six months.

Then in the period which begins in March, 1955—this is one year before he perished—he began to suffer from a series of attacks which were marked by respiratory symptoms—cough, dyspnoea and fever. He had "five or six" of these attacks, and either with the aid of rest or with the passage of time, perhaps encouraged by antibiotics, these attacks subsided; they were accompanied in some instances by a radiological shadow at the right lung base. I shall ask Dr. Calov to comment on some of the films selected from the numerous X-ray films taken during the patient's illness. The chest film taken in July, 1955, was made during one of the more severe "attacks".

DR. W. L. CALOV: Adhesions at the right costo-phrenic angle and also the damage to the ribs are the result of the lobectomy. There are increased lung markings on both the left and right sides, but the nature of the lesion is difficult to determine.

DR. HALL: Evidently those X-ray films did not tell whoever was looking after the patient very much about it, because he was treated conservatively. In January of this year he started out on what sounds like a similar attack to those he had suffered in the previous three or four months. This one, however, did not respond to antibiotic treatment; so that he was admitted to hospital, and apart from having lost a stone in weight, on his admission he complained of no new symptoms.

However, physical examination at this time revealed consolidation at the left lung base, with a pronounced friction rub; his temperature was 101° F., but no other abnormalities were noted on physical examination.

At this stage certain special investigations were performed, which we shall deal with later. During the next six weeks, which bring us out of January into February, he suffered from severe pleural pain, which is described as agonizing, together with a friction rub, and this pain occurred from time to time at both lung bases; there is no consistency as to which side is involved at this stage, and an irregular and persistent fever of up to 103° or 104° F. accompanied by drenching sweats and occasional rigors was recorded. There is another episode which is perhaps not surprising in view of all this—he brought up a little blood-stained sputum. He was given cortisone without any obvious effect, and again further special investigations to be discussed later were performed. The last clinical feature of the story began in March of this year, one week before his death, when he complained of pain and tenderness in the right hypochondrium accompanied by slight jaundice and hepatomegaly, and this clinical change was again investigated with results which we will later hear; then he passed into a semi-comatose state, dying a week after the onset of jaundice.

So the clinical story is one of a period of well-being following lobectomy and the distressing return of respiratory symptoms, which, however, responded to conservative measures; these attacks were accompanied by pleurisy, and sometimes by indefinite and apparently evanescent radiological changes. Then there followed a severe attack of much the same type as initially, but so severe that it did not respond to simple measures, involved admission to hospital, and was accompanied by signs of consolidation at the lung base opposite to that at which the carcinoma had originally presented. Loss of considerable weight was observed, and thereafter a downward path, characterized by respiratory symptoms and fever together with a terminal episode involving jaundice, brought the history to a close.

Now the special investigations. The most striking of these are in the blood picture, as we see clearly set out in the protocol, but I think it may be instructive to revert to absolute values for the white cell counts; otherwise much

TABLE I.

Investigation.	Date and Result.						
	February 2, 1956.	February 9, 1956.	February 16, 1956.	February 27, 1956.	March 8, 1956.	March 15, 1956.	March 26, 1956.
Blood count:							
Haemoglobin value (grammes per centum)	15.3	—	—	—	12.5	11.5	11.6
Leucocytes (per cubic millimetre)	39,000	28,000	38,000	81,000	41,500	95,000	110,000
Neutrophilic cells (per centum)	69	74	86 (Band forms, 10; seg- mented forms, 76)	92 (Young, 2; band, 12; seg- mented, 78)	61 (Band, 13; segmented, 48)	59 (Band, 23; segmented, 36)	66 (Band, 3; seg- mented, 63)
Lymphocytes (per centum)	16	8	5	3	3	1	3
Monocytes (per centum)	6	3	6	3	2	1	2
Eosinophilic cells (per centum)	9	15	3	2	34	39	28
Platelets	—	—	—	286,000	—	59,000	Nucleated red blood cells: one per 100 leuco- cytes.
Erythrocyte sedimentation rate (millimetres in one hour)	14	—	11	—	—	—	—
Other tests	Sputum exam- ination: No cancer cells.	Mantoux test: Negative (1:1000). Scalene lymph node biopsy: No abnormal- ity. Marrow biopsy: See case history.		Stool examina- tion: No ab- normality. Muscle biopsy: No abnormal- ity. Lymph node biopsy: No abnormality.	Serum calcium content: 9.4 milligrammes per centum. Serum phos- phorus con- tent: 3.6 milligrammes per centum. Serum alkaline phosphatase content: 11 King - Arm - strong units.	Zinc sulphate turbidity: 10 units. Serum bilirubin content: 0.2 milligramme per 100 milli- litres. Serum alkaline phosphatase content: 22 King - Arm - strong units. Thymol tur- bidity: "+". Serum protein content: 8.6 grammes per centum. Prothrombin index: 80%. Microscopic urine examina- tion: No abnormality. Casoni test: Negative. Wassermann and Kline tests: Negative.	Stool examina- tion: No ab- normality. Blood culture: Negative. Liver biopsy: See case history.

of the data they provide will be lost. We find that neutrophils range in number from 21,000 to 75,000 per cubic millimetre, so that there is a well-marked neutrophilic leucocytosis. The next most striking change in the blood count is to be found in the eosinophil counts, which vary between 1300 and 38,000, so that there is a marked eosinophilia. Lymphocytes are essentially within normal limits, except in the first count, where they are somewhat raised, but this is not seen in the later counts. Monocytes are also raised, and basophils are absent throughout. The haemoglobin value was normal at first, but there was a slight terminal anaemia, again not very striking. Platelets show a normal count until just before death, when there is a fall, and the sedimentation rate was normal earlier on, but the examination was not repeated. So that in the blood count we have a neutrophil leucocytosis and an eosinophilia.

The next investigation of interest is the marrow puncture. This apparently caused the pathologist some doubt, and he suggested either chronic myeloid leukaemia or a leukaemoid reaction as best describing the picture presented.

Now the liver function tests performed at the time of the episode, already referred to, which involved pain in the right hypochondrium, hepatomegaly and jaundice, revealed evidence of non-specific liver cell damage. The tests certainly do not support the idea that the jaundice was obstructive, and there is no evidence to show that it was haemolytic.

Then there are a series of negative or normal investigation results—stool examination, microscopic examination of urine, blood culture, Casoni, Wassermann and Kahn tests, serum calcium and phosphorus determinations, two lymph node

biopsies, a negative Mantoux reaction soon after admission and a normal muscle biopsy finding.

Now to take this information bit by bit and to ask ourselves how we can account for it. I think it is fair to say that if one were to give these facts to a clinician, he would agree that the probable diagnosis would be carcinomatosis. I imagine that this occurred to everyone who looked after this patient. I imagine it occurred to everyone here, and I suppose a student would be "ploughed" for not mentioning it in an examination. This diagnosis would explain the evolution of the story before operation, and its subsequent recurrence, leading to his death two years after the initial symptoms. This opinion is in no way threatened by the nature of the growth which was anaplastic and squamous. The respiratory symptoms when they returned would suggest perhaps a local extension and recurrence, and the final downward path with recurrent respiratory symptoms would all fit into the picture of carcinomatosis. However, to my mind, there stands in the way of carcinomatosis one great obstacle, and it is the episode which involved the liver. Carcinomatosis could cause jaundice, hepatomegaly and pain in the right hypochondrium. But we have a liver biopsy, the report of which is before us: "Normal architecture; many neutrophils are present in the portal tracts and there are a few small foci of parenchymal degeneration. There is a non-specific hepatitis present." Now in the ordinary course of events carcinoma of the liver may take the form of a discrete deposit varying in size from that of a pinhead to two shillings. It is quite possible that someone performing a liver biopsy under these conditions might miss a nodule and obtain a piece of normal liver. We have all seen patients who have subsequently perished of carcinoma involving the liver, in whom a liver biopsy has revealed normal liver. On the other hand, carcinoma may be seeded throughout the

some part of the biopsy. But, and I am going to ask Dr. Billington his view on this, this biopsy report is not what one would expect in carcinomatosis involving the liver. I wonder if Dr. Billington would like to say something about the liver picture including the liver function tests and the liver biopsy.

DR. B. P. BILLINGTON: With regard to the question of secondary carcinoma of the liver, if we take all the primary sites of malignant disease which give rise to prominent hepatic metastases in certain parts of the world it would be found that bronchial carcinoma is the commonest. I do not think that state of affairs exists in this country, certainly not at the moment. The liver function tests in this instance, to my way of thinking, do not support a diagnosis of secondary carcinoma involving the liver. Either we could expect no abnormality in the tests, or an elevation in the serum bilirubin level, which is sometimes preceded by a rise in the serum alkaline phosphatase levels and bromsulphthalein retention, neither of which is present here. We do have, however, an elevation of the zinc sulphate turbidity level, demonstrating therefore a rise in the γ globulin fraction of the plasma, which may represent an infective element in the story.

Commenting in abstract on the liver biopsy findings, in secondary carcinoma of the liver we would expect to find either normal liver or secondary carcinoma in the liver biopsy specimen. In this instance we find something different—namely, accumulation of neutrophils in the portal tracts with patches of focal necrosis, which I would take to suggest an infective process somewhere, with some reaction to it in the liver. Admittedly, from the clinical point of view, he had a high fever and a leucocytosis, and this might swing one in favour of an infective condition, but this picture of a swinging fever and a leucocytosis may be caused by secondary deposits in the liver. Under these circumstances I do not think that the hepatic story makes the primary diagnosis any clearer. These liver findings as they present themselves would swing me towards an infective cause, although they do not generally fit the general downward course of the patient's illness, with secondary deposits from bronchial carcinoma in the background.

DR. HALL: Bearing in mind the possibility of carcinomatosis, it remains to consider what conditions other than carcinoma could explain this, and this consideration involves postulating two separate diseases. The first disease, which on the clinical picture alone warrants very definite mention, is pulmonary tuberculosis. It would explain the history of the recurrence after operation. Tuberculosis may have been dormant at the time of operation. It would explain the respiratory complication which ushered in the patient's last illness. It would explain the high fever, and it might even have been activated by two weeks of cortisone therapy. It would explain the whimsical lung picture, sometimes on the right and sometimes on the left. If it involved miliary spread, it might explain jaundice, hepatomegaly, pain in the right hypochondrium and the picture of neutrophil leucocytes invading the portal tracts of the liver. The Mantoux reaction was negative about the time that second X-ray film was taken, but in miliary tuberculosis this finding is possible.

A third group of conditions which I think the clinical story might bring to mind are the collagen diseases, especially polyarteritis nodosa. There is very little positive clinical evidence to support them. They would explain the fever and the terminal episode involving the liver. They would explain the respiratory symptoms, and they might even explain in some way the appearance seen in the lung X-ray films; and perhaps they are made more probable by certain special investigations which we will mention later.

Fourthly, I think that certain conditions involving sepsis, certain fevers and infective processes, could be grouped together and mentioned at this point. Bacterial endocarditis is suggested by high fever, wasting and a fatal termination. However, there is nothing in the history to suggest embolic phenomena, unless the episodes in the chest were embolic and the subacute bacterial endocarditis involved the right side of the heart. We have no clinical evidence of heart disease from which such emboli would arise, but it remains a possibility which I think should be excluded when we come to discuss the special investigations.

Typhoid fever and brucellosis always pass through a clinician's mind when he is unable to explain a fever, but here again there are no symptoms which specifically point to either of these maladies.

Fungal diseases, I suppose, to get on to rarer conditions, should be mentioned, but again cannot be supported by clinical findings.

Pus hidden away somewhere, for example, under the diaphragm, might, had the story been timed a little differently, have been a possibility especially if the return of respiratory symptoms had followed the operation sooner than they did. Had the patient not made such a splendid initial recovery, then we might have been justified in thinking of pus either above or below the diaphragm. This would explain the leucocytosis and some of the other special investigations, but it seems unlikely with such a long latent interval.

We have no reason to suspect pyelonephritis, which I suppose is a well-known trap at clinico-pathological meetings when fever is a feature of the story. Bowel infestations or primary liver disease are supported neither by the clinical story nor by the special investigations.

Now, if we pass from the clinical picture to the information derived from the special tests, certain causes of neutrophil leucocytosis might come to mind, and I would like to recall those which I think are relevant to this patient—tuberculosis, malignant disease (which can both cause a considerable neutrophil leucocytosis), leucemia and the collagen diseases. Sometimes it is the effect of certain drugs, which I think we can eliminate in this case—the patient was an electrical fitter, and apart from "sulpha" drugs there is no evidence that he was taking any drugs which are likely to cause such a marked leucocytosis as this.

Eosinophilia suggests a number of possibilities, some of which are applicable to this case. Parasites—we have no knowledge of this man's background, whether he lived abroad, and no evidence to support any of the common parasitic conditions that lead to eosinophilia. Allergic conditions—there is nothing in the way of skin rashes or in the previous history to support this possibility. Myeloid leucemia can sometimes be associated with an eosinophilia. Collagen diseases—and I suppose this is the one real point which makes collagen diseases worth discussing in this case—must again be considered in spite of the fact that the bone X-ray films were normal. And finally, Hodgkin's disease must be mentioned.

Finally, the third piece of information available from the haemopoietic system was the marrow picture. From the clinical point of view we rule out chronic myeloid leucemia—there were two normal lymph node biopsies, hepatomegaly was very decidedly terminal, and there was no splenomegaly. The neutrophil count is not made up of immature cells suggesting leucemia. The whole evolution of the illness, I think, counts strongly against this disease. Thus we can rule out myeloid leucemia and discuss the causes of a leucemoid reaction of the bone marrow, which in retrospect seems the more probable of the two suggestions offered by the hematologist.

The causes of a leucemoid picture include carcinoma involving bone, tuberculosis, and certain drugs (such as "sulpha" drugs); but I think on the whole that drugs can be omitted from further consideration; and then multiple myeloma, against which normal bone X-ray films should be mentioned. And in addition, osteosclerosis, Paget's disease, and myelosclerosis are all unlikely on the same grounds. Hodgkin's disease is another possible cause of a leucemoid bone reaction.

To take these suggestions in turn, bearing in mind the clinical story, I think that miliary tuberculosis must be retained as a possibility. It would account for the neutrophil leucocytosis, though, as I have said, that picture is unusual. Sometimes a neutropenia is seen. It has been known to cause eosinophilia (although this is rare), and it can cause a leucemoid bone reaction. I have seen autopsy specimens from a case in this hospital, in which a leucemoid bone reaction and a leucocytosis occurred early, leading later to a leucopenia and anaemia, and finally a leucocerythroblastic anaemia, but, without eosinophilia.

Carcinomatosis appears on every count—special tests and clinical story—to be possible. It can certainly cause a neutrophil leucocytosis, although one might consider the present levels very high. It can sometimes cause eosinophilia, although not a common cause, but certainly a possible one, and it can certainly cause a leucemoid bone marrow. It will at once be objected that the bone X-ray films were normal: the spine and pelvis were submitted to X-ray examination at a time when this blood picture was to be seen. However, in certain museums of teaching hospitals in London there are specimens of bones which the pathologist has mounted very beautifully, and they are studded with carcinoma; beside them are X-ray films taken a day or two before death, yet showing no evidence of liver, in which event malignant cells would be expected in

carcinoma at all. So on that account I feel that this still remains the most probable clinical explanation for this story.

Hodgkin's disease to my mind is most unlikely. Two gland biopsies were found to be normal. Lymph glands were not described as palpable. The hepatomegaly was terminal, the spleen was not enlarged, and no other features of Hodgkin's disease were present, except perhaps fever. On these accounts I would discard Hodgkin's disease.

I think allergic diseases must be ruled out. If parasites caused this, we just have not sufficient information at hand to arrive at the correct diagnosis. Collagen diseases, I think, still remain possible.

To review the situation at this stage: miliary tuberculosis, carcinomatosis and collagen diseases must still be included.

On clinical grounds, in addition to this, we could mention subacute bacterial endocarditis, although we have been sadly misled if that were the case. And so I come to the consideration of the above three possibilities.

If tuberculosis is the cause of this condition, then presumably it is miliary tuberculosis, and I regard this, taking into consideration perhaps that this is a clinico-pathological conference and we do not expect things to be as straightforward as they might appear, as the most likely explanation for all these signs and symptoms; and I think it explains the liver story much more adequately than does carcinomatosis. Carcinomatosis I put second, although only just second. And if it were carcinomatosis then it is presumably a later extension and distant metastases of the original carcinoma. It seems improbable to me that this carcinoma could have come from somewhere else in the first place, the pulmonary lesion being secondary. In the case of a squamous, anaplastic carcinoma one would have expected to find evidence of the primary carcinoma at the time of operation in, say, the mouth or anal canal. So that if this is a carcinoma, it presumably represents metastases and extension from the initial pulmonary lesion.

Thirdly, collagen disease with *periarteritis nodosa* is the most probable of these and not very probable at that. It is mainly, I think, on account of the eosinophilia that one is inclined to mention that possibility.

So I would conclude by considering the three possibilities to be (i) miliary tuberculosis, (ii) carcinomatosis, and (iii) *periarteritis nodosa*, in that order.

Dr. E. H. STOKES: This case is a very difficult one, and I congratulate Dr. Hall on the logical way in which he has approached it. Now first of all, miliary tuberculosis, although he has it number one on his list, does not sound to me very striking, especially with those X-ray films. Then you have the negative Mantoux reaction, which, as you know, you may get in miliary tuberculosis as an anergic reaction, but I really cannot see any evidence favouring miliary tuberculosis. The positive things that we have are the fact that there was a carcinoma at the hilum of the right lung, and the deposits there in the left lung later on. The leucæmoid reaction is difficult to explain, but it may result from the carcinomatosis.

In *periarteritis nodosa* you certainly may get an eosinophilia. Taken all in all, I would think that carcinomatosis is the most likely diagnosis. Dr. Hall mentioned brucellosis, but you must remember that he mentioned it only in passing. Brucellosis is a disease one has to be on the lookout for. One finds it usually in people who are associated with cattle. It is a common disease in America in the big meat-processing places. It is not very common in Australia, but sometimes one gets a case. I had a case two years ago who fortunately responded to "Aureomycin". With regard to brucellosis, you get a continued fever, you get an enlarged spleen, and you get malaise and loss of weight. These are the usual clinical phenomena of chronic brucellosis. Finally, in conclusion, I favour the diagnosis of carcinomatosis.

Dr. R. WALSH: The only comment which I have to offer on the blood picture is that in a leucæmoid reaction I would have expected considerably more immature cells than are listed here. There is a very high leucocytosis, 95,000 and 110,000 per cubic millimetre, with only 3% and 23% band forms respectively in the two counts. It is also unusual to find relatively few nucleated red cells in such a leucæmoid reaction or leuco-erythroblastic anemia due to carcinomatosis. I was wondering whether Dr. Hall would comment on the rather high protein figure of 8.6 grammes per centum.

Professor K. INGLIS: Might I suggest that possibly infection was superadded to the malignant process in the lung, and that that had become widespread, and that the high leucocyte count and the reaction in the bone marrow might have been the result of infection? Dr. Stokes emphasized

carcinomatosis, but I would suggest for consideration that an infective element was superadded and accounts for some of the features.

Dr. E. HIRST: I would like to make an altogether different diagnosis, that of bilateral pulmonary eosinophilic infiltration (Löfller's syndrome). Of course, this diagnosis ignores the findings relevant to the liver, which I find on all counts extremely puzzling. Firstly, the investigations are given as of March 15, whereas the protocol states that symptoms and signs of hepatic disease were present on March 19. Assuming then that investigations were made after the onset of hepatic symptoms, I cannot understand how anyone can show clinical jaundice with a serum bilirubin of 0.2 milligramme per 100 millilitres. The alkaline phosphatase and high serum protein figures are to me equally difficult to interpret. In view of these conflicting features I suggest that the hepatic disease was a hepatitis at most, and was not related to the main clinical picture which implicated the lungs and the bone marrow.

The outstanding features to me then are the continuous pulmonary signs and the leucocytosis, especially the notable eosinophilia.

I do not care for the suggestion of carcinomatosis. He may indeed have had carcinomatosis, but then why are there not adrenal or cerebral metastases, as is usual?

Apart from the pulmonary signs there is the eosinophilia. Of course, one could explain it by Hodgkin's disease or periarteritis, but there is nothing whatever to substantiate these diagnoses.

A diagnosis which could explain both the pulmonary signs and the marrow response is pulmonary eosinophilic infiltration, and that is what I suggest this patient had.

Dr. STOKES: May I ask a question of Dr. Hirst? Do they usually die of Löfller's syndrome?

Dr. HIRST: I do not know. This patient did.

Dr. A. A. PALMER: May I make a comment? I am most intrigued with this extremely high eosinophilia. I am completely puzzled. The only thing I can think of to explain it is that in some squamous carcinomas you do find, quite rarely, a high local concentration of eosinophils, and I wonder whether this is such a squamous carcinoma. Perhaps if it is diffusely spread in many parts of the body it might induce an eosinophilia. If it is carcinomatosis, as Dr. Hirst has mentioned, the spread is unusual. We do, of course, sometimes get a diffuse spread, which would not show in the X-ray film any more than is seen in this case.

Dr. G. A. W. JOHNSTON: The first white cell count of 29,000 per cubic millimetre without a shift to the left is very interesting; also is the fact that the blood sedimentation rate is not significantly raised. When presented with such findings, one immediately thinks of an infiltrative lesion of the bone marrow. In subsequent blood counts there is a shift to the left, and nucleated red cells appear in the peripheral blood. I find it difficult to understand why an infiltrative lesion of bone marrow causing this degree of disturbance is not also associated with a more severe depression of haemopoiesis. Perhaps the picture is masked by cortisone therapy, which may make the diagnosis of leucæmia very difficult. I do not think this diagnosis of leucæmia can be so easily excluded in this instance. With regard to the high serum protein level, it is quite possible that this patient may have produced abnormal proteins such as cryoglobulin or macrogamma globulin (as a result of liver disease).

Dr. B. P. BILLINGTON: May I ask a general question? If we explain this case on the basis of carcinomatosis, how do we explain the episodes of pulmonary involvement which came and went? It appears clear from the films that the changes not only appeared but disappeared repeatedly. It seems much more like a story of recurrent pulmonary infarcts, due either to emboli or to local thrombosis. Further, with respect to the liver, the thing that strikes me as peculiar if the disorder is explained on a secondary neoplastic basis is that one day no one noticed the liver, and the next day it was painful, tender and four centimetres enlarged.

Dr. W. L. CALOV: I do not propose to enter at length upon the discussion of this case, but I would like to say that carcinomatosis would fit the picture, whether there was superadded infection as Professor Inglis suggested or not. The recurring bouts of pain I think could still be explained on the grounds of carcinomatosis. There could be seedings on the pleura, and there could be bleeding. Now, blood in the pleural space causes a lot of pain, in my experience of it. In the old days, when we used to do quite a number of

artificial pneumothoraces and we split blood in the pleura, it used to hurt. I think that this pain can be explained on those grounds. I would say that carcinomatosis is the most likely diagnosis. I say this not on any scientific basis, but because it seems to me to be the common-sense view. The fellow had a lobectomy for a carcinoma, and some time later he died, and the most likely thing is that he died of the carcinoma.

Dr. Rose: Dr. Hall, have you anything to say in conclusion?

Dr. Hall: I would like to say that I came here tonight not with a very well-formed idea as to whether I should put miliary tuberculosis or carcinomatosis first in the final diagnosis, and I think this doubt was increased by the X-ray films which I had not seen before. I feel that as three senior physicians have voted for carcinoma, I shall reverse the order by putting carcinomatosis first, miliary tuberculosis second, and leaving collagen disease a very poor third.

I cannot explain the raised protein level, which might be accounted for by *periarthritis nodosa*, but not by either of the first two conditions. I think that with one single reading we must regard the protein figure as inexplicable in the light of the findings.

I think that Dr. Billington's comments on the liver are very pertinent, and they made me think of tuberculosis first, and carcinomatosis second, and I still cannot explain his objection. As to the lung picture, I take it that recurrent emboli could occur from carcinoma. They may come from deep veins perhaps being pressed upon by metastases elsewhere. To object to carcinomatosis on the grounds of spread, and this spread is unusual, only makes me more inclined to put carcinoma first, because unusual spread is something which has no meaning in carcinomatosis—it can go anywhere, and it does go anywhere. On common-sense grounds I agree—carcinomatosis is much more likely. It is more common than miliary tuberculosis. As regards Löfner's syndrome, it is to my knowledge a benign condition, which is self-limiting, and I have never read of patients dying of this disease. In spite of the unusual blood picture, and it is unusual for any of the conditions which are suggested, I would offer as my final choice: (i) carcinomatosis, (ii) miliary tuberculosis.

Report of Autopsy.

PROFESSOR F. R. MAGAREY: I am going to try and show that these varied clinical manifestations are all, in a rather roundabout way, the result of carcinoma.

Firstly, sections of the tumour removed at operation show it to be of the large cell, anaplastic type, so that there is no doubt about the original diagnosis of carcinoma of the lung. The pulmonary symptoms, which formed such a large part of the later clinical stages, are not the result of local recurrence of the tumour, but rather are the effects of a series of pulmonary infarcts, which will be explained later on.

Secondary carcinomatous metastases could be found only in the suprarenals—a small one in the left and one much larger in the right. This deposit in the right suprarenal was mostly necrotic and lay very close to the inferior vena cava, which at this level was almost filled with pale friable thrombus, thrombosis presumably having spread via the right suprarenal veins. The caval thrombus extended upwards past the level of the ostia of the hepatic veins, occluding some.

Macroscopically the right side of the liver was dark red and mottled, the left half appearing normal. Histologically the affected portion showed some centrilobular zonal necrosis and congestion, whilst there is a good deal of polymorphonuclear infiltration of the portal tracts. Presumably the liver biopsy was taken from this area, and I believe that most of the change is due to hepatic venous obstruction.

Emboli had broken free from the proximal end of the caval thrombus and had become lodged both on the wall of the right ventricle and in the pulmonary arteries. In both places, wherever clot had adhered, carcinoma had commenced to grow. Histological sections of clot showed it to contain tumour cells, so that the emboli had seeded cancer, which had indirectly metastasized from the secondary deposits in the suprarenal; one might call these tertiary deposits. The pulmonary emboli had caused the formation of multiple infarcts of varying ages scattered throughout the lungs.

There is left to be explained the fever, leucocytosis and eosinophilia. No metastases were found in the bone marrow, and there was no evidence of leucæmia at autopsy.

It is thought that these manifestations can be explained by the various necrotic processes. There was the large necrotic suprarenal tumour, the necrotic blood clot in the inferior vena cava, right ventricle and pulmonary arteries; also a good deal of hepatic necrosis, which has been already described; and finally much pulmonary necrosis resulting from the multiple pulmonary infarcts. Leucocytosis and fever can be explained on this account in much the same way as they can result from myocardial infarction in hypernephroma. It is suggested that the eosinophilia is the result of autosenesitization of the patient to his own degenerating tissues by some sort of hypersensitivity or immunological mechanism which I, for one, do not understand.

Diagnosis.

Secondary carcinoma of the suprarenal glands and liver with thrombosis of the inferior vena cava and recurrent pulmonary emboli.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

MAINTAINING ORDER AT NORFOLK ISLAND.¹

[Extracts from Orders issued at Norfolk Island by Lieutenant-Governor P. G. King, November, 1791.]

THE Inhabitants will sweep round their Huts and keep the Streets clean. If any Filth or other Nuisance is Suffered to gather in or about their Huts, the Inhabitants will for the first Offence work on Saturdays, and for the Second will be turned out of their Huts and other people be put into them. All Swine which are found stragling [*sic*] about near any Cultivated Grounds will be Seized and made use of for the Publick. Fish and Fowls to be cleaned at the seashore.

Every person who is convicted of Profane Oaths or any Scandalous and Indecent Words will on conviction before one of His Majesty's Justices assigned to keep the peace, receive such punishment as they may deserve. Every woman convicted of Blasphemous or Indecent Language will for the First Offence have her hair cut off, for the Second she will be shaved, and for a Third Offence will be whipped at the Carts Tail.

Special Correspondence.

PARIS LETTER.

FROM OUR SPECIAL CORRESPONDENT.

A Surgical Approach to the Upper Part of the Abdomen.

EVEN with resection of the xiphoid process, the surgeon does not find it very easy, if he makes his approach through the normal median incision, to work on the upper part of the stomach, including the cardia, the abdominal part of the oesophagus and the coeliac region. That is why many surgeons prefer a thoracotomy for performing, for example, oesophago-ileostomy after total gastrectomy. Examination of a lateral skiagram shows how deep and difficult the approach to the cardia can be. If a line is drawn from the middle of the median incision to the cardia, it is more or less long and oblique, according to the patient's morphology. To make it shorter and vertical, the only possibility is to cut the sternum. After that, it is necessary to incise the front part of the diaphragm up to the pericardium and to move aside the left lobe of the liver.

A technique of sternotomy devised several years ago by Paul Orsoni and Marcel Lemaire² is still fairly new, even in

¹ From the original in the Mitchell Library, Sydney.

² *Semaine Méd. Paris*, November 18, 1953.

France where it originated. The median incision of the skin is continued up to the fourth or fifth intercostal space. On each side of the sternum one must find the first intercostal space that is really a space—in other words, a break between the cartilages. It is the fifth in broad men, the fourth in thin men. The sides of the sternum are stripped or cleared, care being taken to avoid the internal mammary artery by keeping close to the bone. On the posterior surface of the xiphoid process, the knife or scissors penetrates close to the bone in front of the peritoneum which goes up to cover the diaphragm. After that it is easy to push aside gently with a finger the insertions of the diaphragm on each side, and also the pericardium and the pleural cul-de-sac. The sternotomy itself is now possible without any danger. The instrument required is simply a good costotome like the Doyen costotome. The transverse section is made first, at the level of the fourth or fifth intercostal space that has been previously cleared. Then the vertical section follows, giving to the sternotomy the shape of a "T". A strong retractor placed between the two halves of the sternum pushes them to each side. A median phrenotomy opening is made up to the pericardium, usually about two inches long. If the pericardium is opened, which does not matter, its suture is very easy. Now the surgeon faces the upper surface of the left lobe of the liver and the triangular ligament. This ligament is cut from left to right up to the falciform ligament, and then the left lobe of the liver can be folded towards the right. The whole region of the cardia and the abdominal part of the oesophagus are perfectly exposed and accessible.

With this technique the sternotomy itself can be performed in a few minutes, with insertion of a few haemostatic ligatures at the back of the xiphoid process and during the phrenotomy. The only risk is in the opening of the pleural cul-de-sac. For that reason it is advisable to operate with the patient under anaesthesia associated with tracheal intubation. Repair of the sternotomy wound is simple; suture of the phrenotomy wound from the depth to the surface is followed by suture of the peritoneum and suture of the two parts of the bones or simply of the fascia. Surgeons who are familiar with this procedure state that it is often extremely useful in obtaining wide, easy and comfortable access to the deep celiac region.

Correspondence.

THE WONDER DRUGS OF PSYCHIATRY.

SIR: The above heading was used by me to wonder about the "ataraxics" (M. J. AUSTRALIA, July 14, 1956). After a gestation of my question for three months Dr. Douglas Everingham says, "I do not pretend that new drugs have cured or explained anything" (M. J. AUSTRALIA, August 25, 1956), which, of course, is my contention. *Ne sutor ultra crepidam.*

Yours, etc.,

607 New South Head Road,
Rose Bay,
New South Wales.
October 28, 1956.

GODFREY HARRIS.

NOMENCLATURE AND DRUGS OF THE CORTISONE GROUP.

SIR: I wish to protest strongly through these columns concerning the multitude of names being designated by drug companies to the various adrenocortical hormones and their derivatives.

It appears we have four basic substances, cortisone, hydrocortisone, prednisone and prednisolone, in order of their discovery and approximate strengths. These agents have been given such trade names as "Cortisyl" and "Cortone" for the cortisone compound; "Hydrocortisyl", "Hydrocortone", "Cortril" and "Cortef" for the hydrocortisone compound; "Decortisyl", "Deltacortone", "Meticortin" and "Deltacortilan" for the brands of prednisone; and "Codelcortone", "Deltacortril", "Deltacortef" and "Deltastab" for brands of prednisolone, to mention a few.

There appears to be no uniformity such as prefixing "delta" *et cetera* to any of the above names, and it can be imagined how confusing these drugs are to medical and nursing staff, particularly as dispensers themselves are often

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 27, 1956.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2(1)	4(2)	6
Anthrax	2
Ancylostomiasis	1	..	8	9
Brucellosis
Cholera
Chorea (St. Vitus)
Dengue
Diarrhoea (Infantile)	5(3)	18(17)	2(2)	25
Diphtheria	1(1)	..	1(1)	2
Dysentery (Bacillary)	1(1)	2(1)	..	1	..	1	..	5
Encephalitis	3(3)	..	2	6
Epilepsy
Homologous Serum Jaundice
Hydatid	1(1)	1
Infective Hepatitis	108(34)	37(14)	..	4(4)	1	9	1	..	160
Lead Poisoning
Leprosy
Leptospirosis	1	3
Malaria	2(2)
Meningococcal Infection	2(1)	3(1)	3(1)	..	2	10
Ophthalmia
Ornithosis
Paratyphoid
Plague
Poliomyelitis	1	12	2	..	3	18
Puerperal Fever
Rubella	59(32)	2(2)	20(16)	1(1)	..	2	..	78
Salmonella Infection
Scarlet Fever	12(8)	13(11)	4	..	1(1)	30
Smallpox
Tetanus	2	2
Trachoma	1(1)	..	35	..	36
Trichinosis
Tuberculosis	28(19)	10(8)	9(1)	11(5)	12(6)	7(3)	..	2	77
Typhoid Fever	1(1)	2(2)	3
Typhus (Flea-, Mite- and Tick-borne)	3	3
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

in doubt as to which trade name represents what. As a result, on several occasions I have known patients to be given a wrong substance and consequently incorrect dosage.

Before we are inundated with a variety of other confusing names relating to these products, and before tragic results due to mistakes in prescribing start occurring, I would like to express a plea that drug companies be approached and asked to label their products with basic names.

The Morris Hospital,
Northfield,
South Australia.
October 22, 1956.

Yours, etc.,

R. MUNRO FORD, M.D.

PHARMACEUTICAL BENEFITS AND THE TREATMENT OF LEUCHEMIA.

SIR: I wish to make a plea that the drugs cortisone, 6-mercaptopurine and "Myleran", which are used in the treatment of leuchæmia, be included in the pharmaceutical benefits list. These drugs are beneficial in the relief of symptoms, and with their aid patients are able to carry on normal activities. In several instances men have worked at their jobs for periods of more than twelve months without the loss of a day's work from illness—a record which is frequently better than the patients' fellow workers. Sir Lionel Whitby and Professor Witts, who have recently lectured in Melbourne, are both agreed that the treatment of leuchæmia with these drugs is worth while, and in the acute form of the disease a remission can be obtained in the majority of cases. I have written to the Director-General of Health and urged that these three drugs be included in the pharmaceutical benefits list and would be pleased if further support could be given to this request.

Yours, etc.,

JOHN A. McLEAN.

417 St. Kilda Road,
Melbourne,
October 24, 1956.

Royal Australasian College of Surgeons.

FINAL FELLOWSHIP EXAMINATION.

THE next meeting of the court of examiners for the final examination for Fellowship of the College will be held in Melbourne, beginning on Friday, May 3, 1957. Candidates who desire to present themselves at this examination should apply, on the prescribed form, to the Censor-in-Chief for permission to do so before March 21, 1957. The appropriate forms are available from the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne.

Candidates who have already been approved by the Censor-in-Chief, but who have not yet presented for the examination, may present at this examination, provided they notify the Secretary of their intention to do so by March 21, 1957. It is stressed that entries close on this date and that late entries cannot be accepted.

The examination fee is £21, plus exchange on cheques drawn on banks outside Melbourne, and must be paid to the Secretary by March 21, 1957.

The examination will be conducted in general surgery and in the special branches of ophthalmology, laryngology, gynecology and operative obstetrics, orthopedics, urology, neurosurgery, plastic surgery and thoracic surgery.

At its meeting held on June 23 and 24, 1956, the Council decided that until December 31, 1958, Fellows of other Colleges with which the Royal Australasian College of Surgeons has reciprocity of primary examinations, who obtained their Fellowship prior to January 1, 1950, may, at the discretion of the Council, be permitted to undergo a modified type of final examination. The conditions set out above regarding method of application for permission to present, date on which entries close, examination fee et cetera also apply to the modified type of examination.

Nominations and Elections.

THE undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Forbes, James Ian, M.B., B.S., 1955 (Univ. Adelaide), 60 Halton Terrace, Kensington Park, South Australia.

The undermentioned has been elected as a member of the South Australian Branch of the British Medical Association: Materne, Maurice Ernest, M.B., B.S., 1955 (Univ. Adelaide).

Deaths.

THE following deaths have been announced:

PHILLIPS.—Doreen Guinness Philipps (Viscountess St. Davids), on October 19, 1956, at Kew, Victoria.

ABBOTT.—Joseph Henry Abbott, on October 31, 1956, at Sydney.

GRIFFITHS.—William James Griffiths, on November 5, 1956, at Queanbeyan, New South Wales.

Diary for the Month.

- Nov. 19.—Victorian Branch, B.M.A.: Finance Subcommittee.
- Nov. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
- Nov. 22.—Victorian Branch, B.M.A.: Executive of Branch Council.
- Nov. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
- Nov. 23.—Queensland Branch, B.M.A.: Council Meeting.
- Nov. 27.—New South Wales Branch, B.M.A.: Ethics Committee.
- Nov. 28.—Victorian Branch, B.M.A.: Branch Council.
- Nov. 29.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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